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Table of Contents

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ORIGINAL ARTICLES— PAGE.
"The Anæmias", by WILFRED EVANS, M.B., M.R.C.P. 331
"The Infection of the Placenta and of the Fœtus by Induction of Labour in Eclampsia", by W. J. PENFOLD, M.B., Ch.M., D.P.H., B.Hyg., M.R.C.S., L.R.C.P., and HILDRED M. BUTLER, B.Sc. .. 336
"Plumbing", by H. C. RUTHERFORD DARLING, M.D., M.S., F.R.C.S., *et cetera* .. 341
"Nasal Sinusitis in Relation to Pulmonary Tuberculosis", by CLIVE M. EADIE, M.B., B.S., F.R.A.C.S. .. 345

REPORTS OF CASES—
"The 'Doubtful Vertex': Six Cases", by JOHN S. GREEN, M.D., D.G.O. .. 346

REVIEWS—
Biological Chemistry for the Clinician .. 347
Breast Feeding .. 348
The Localization of Disease .. 348

LEADING ARTICLES—
Miners' Nystagmus .. 349

CURRENT COMMENT—
Focal Infective Prostatitis .. 351

ABSTRACTS FROM CURRENT MEDICAL LITERATURE— PAGE.
Therapeutics .. 352
Neurology and Psychiatry .. 353

SPECIAL ARTICLES ON TREATMENT—
The General Management of a Patient with Early Pulmonary Tuberculosis .. 354

CORRESPONDENCE—
The Significance of Oral Sepsis in the Adult .. 355
An Economic Commentary and Diagnosis .. 356
Diathermy of Tonsils .. 357

PROCEEDINGS OF THE AUSTRALIAN MEDICAL BOARDS—
Tasmania .. 357

OBITUARY—
William Bradridge Studdy .. 358

BOOKS RECEIVED .. 358
DIARY FOR THE MONTH .. 358
MEDICAL APPOINTMENTS .. 358
MEDICAL APPOINTMENTS VACANT, ETC. .. 358
MEDICAL APPOINTMENTS: IMPORTANT NOTICE 358
EDITORIAL NOTICES .. 358

THE ANÆMIAS.[1]

By WILFRED EVANS, M.B. (Sydney), M.R.C.P. (London),
Honorary Assistant Physician, Sydney Hospital; Honorary Physician, Lewisham Hospital, Sydney.

I PROPOSE, before considering the ætiology and pathology of the individual types of anæmia, to discuss briefly the physiological principles which underlie blood formation and destruction in the human body in general.

Red blood cells are formed from the endothelium lining the capillaries of the cancellous bones and the ends of the long bones. It should be realized that the vascular system of the bone marrow is continuous with and forms part of the general circulation. From the endothelial cells the megaloblasts, which are the most primitive type of red cells, arise, and from them in succession are formed normoblasts, reticular cells and finally the mature red cells of the circulating blood.

One megaloblast may give rise to several normoblasts, and these are capable of multiplying indefinitely so that the endothelial cells are not under normal circumstances called into great activity. The intermediate stage between the normoblast and mature red cell is called the reticulocyte because with certain stains it exhibits a peculiar network which is the remains of the basophile cytoplasm of the immature cell. The fully developed red cell has no power of proliferation, its average duration of life is thirty days, and it is gradually disintegrated by the force of the circulation. Exercise by increasing wear and tear accelerates the disintegration and so rest is important in the treatment of anæmia.

In considering any anæmia it is of great importance to determine whether the condition is due to increased blood destruction or diminished blood formation. The determination of this is one of the greatest difficulties in the study of hæmatology and we have as yet no completely reliable guide.

It had been hoped that the estimation of the output of bilirubin would be a reliable index of the amount of blood destruction, but further investigation has brought to light certain fallacies which detract from its value. Bilirubin is one of the breakdown products of the hæmoglobin of the red blood cells. It is taken to the liver and excreted in the bile to the intestine, when it forms stercobilin. The amount of stercobilin excreted in the stools is readily estimated, but, unfortunately for this test, bilirubin is not entirely a waste product. A considerable amount of it is reabsorbed into the circulation and used for the formation of fresh blood cells, and so an excess of bilirubin or stercobilin in the stools may mean, not increased blood destruction, but decreased blood formation, because the bone marrow is incapable of utilizing the bilirubin in the manufacture of red blood cells.

Estimation of the number of reticulocytes is much stronger evidence of increased blood formation.

[1] Read at a meeting of the North Shore Clinical Society on December 6, 1932.

When the bone marrow is very active, after hæmorrhage or in acholuric jaundice, the reticulocyte count rises from the normal 1% to as much as 30%, and even more immature cells, normoblasts or even megaloblasts may then appear. Increase in the uric acid content of the urine is also evidence of increased blood formation, as uric acid is largely derived from the loss of the nucleus when the normoblast becomes a mature red cell.

In the classification of anæmias the condition of the bone marrow is of the first importance, and we may divide them into two main groups: (i) those with hyperplasia of the bone marrow—the hyperplastic anæmia; (ii) those with aplasia of the bone marrow—the aplastic anæmias.

Hyperplastic Anæmia.

The hyperplastic anæmias form far the largest and most important group, and are divided into: (*a*) The nutritional group, due to diminished blood formation; (*b*) the hæmolytic group, due to increased blood destruction.

The Nutritional Group of Hyperplastic Anæmias.

The anæmias in the nutritional group develop when the bone marrow is deprived of certain substances which are essential to the development of the mature red cells from the endothelial cells of the bone marrow. These substances are found in the normal human diet, and it is of profound importance to realise that the elements which are essential to the development of the normoblast from the megaloblast are entirely different from those which are necessary for the development of the mature red cell from the normoblast, and the type of anæmia will depend upon the stage at which development is arrested. It will be megaloblastic in type if the substances necessary for the development of the megaloblast to the normoblast are lacking and they are the active principle of liver and vitamin *B*. It will be normoblastic or microcytic in type if the substances necessary for the development of the mature red cell from the normoblast are lacking, and they are vitamin *C*, thyroxin and iron.

Nutritional Anæmias due to Lack of Stomach Ferment.

The most important member of this great group of nutritional anæmias is pernicious anæmia. Until quite recently this disease was always considered to be hæmolytic in origin, and our conception of the origin of this disease has undergone radical changes since the advent of liver treatment.

The bone marrow in pernicious anæmia is enormously increased and extends into the shafts of the long bones. And yet this increase of bone marrow is accompanied by a diminished output of cells. Witts, in his recent article, ably puts it that the condition is analogous to a mass production motor car factory when an essential part is lacking and the assembly hall is crowded with unfinished motor cars. The chief arguments which are advanced to show that pernicious anæmia is of nutritional and not hæmolytic origin are: (i) The improvement which results when liver extract is supplied and the nutritional deficiency made good. It is very difficult to explain the improvement which results in any other way than by replacing a deficient substance. (ii) The fact that the reticulocyte count is low in a case of untreated pernicious anæmia. If it were of hæmolytic origin we would expect a high reticulocyte count in an endeavour to repair the damage. (iii) In the stage of recovery after the administration of liver extract the reticulocyte count rapidly rises even to as high as 30% and following this there is a rapid rise in the number of red cells. Furthermore, the excretion of uric acid (which is due to the loss of the nucleus when the normoblast develops to the mature red cell) rises rapidly during the reticulocyte crisis. This means that development of the red cells to maturity is once again taking place.

Some very important work has recently been done by Castle in elucidating the cause of pernicious and allied anæmias. He has shown that the basic cause of pernicious anæmia is the absence of a ferment in the stomach. It is not the hydrochloric acid or the pepsin of the stomach which is the important factor, but this third substance, which is of a rennet-like nature. This ferment acts on certain food materials and produces a substance which is essential for the

Table I.
Classification of the Anæmias.

- **The Anæmias**
  - **Hyperplastic**
    - **Nutritional (associated with defective blood formation), due to**
      - **(a) Lack of stomach ferment.**
        - **Pernicious anæmia.**
      - **(b) Deficiency in diet.**
        - **Nutritional anæmia of infants.**
        - **Anæmia of scurvy.**
        - **Tropical nutritional anæmia.**
        - **Simple achlorhydric anæmia of middle-aged women.**
        - **Von Jaksch's anæmia.**
      - **(c) Defective absorption from the alimentary tract.**
        - **Tuberculosis of the bowel with resulting stenosis.**
        - **Gastro-intestinal ulceration from chronic dysentery.**
        - **Infection with *Dibothriocephalus latus*.**
        - **Coeliac disease.**
    - **Hæmolytic (associated with increased blood destruction).**
      - **Acholuric family jaundice.**
      - ***Icterus gravis neonatorum*.**
      - **Paroxysmal hæmoglobinuria of syphilis and blackwater fever.**
      - **Acute hæmolytic anæmia.**
      - **Chronic acquired hæmolytic anæmia.**
      - **Hæmolytic anæmia of pregnancy.**
  - **Aplastic**

development of the red blood corpuscles. It was originally thought that the food material upon which it acted was a protein, but very recently Castle has shown it to be vitamin B_2.

Anæmias allied to pernicious anæmia may be produced if the vitamin is absent from the food, as in the tropical pernicious anæmias, or if absorption from the bowel is interfered with as in sprue and cœliac disease. It is interesting to note that these tropical anæmias in some cases have been cured by the administration of vitamin *B* alone given as yeast or mannite. But in pernicious anæmia the ferment is lacking in the stomach, and hence it is necessary to administer liver or its extract or hog's stomach, as these contain the products of the interaction of the ferment and vitamin B_2.

The clinical features of pernicious anæmia and the microscopic appearances of the blood film are well known to you all, and I shall recapitulate only their most striking features.

Clinically one notes the pallid lemon-yellow colour of the skin, the slight icterus of the conjunctivæ, the pale smooth and frequently sore tongue with denuded papillæ. Oral sepsis is common and the patient complains of progressive weakness and breathlessness and frequently of numbness and tingling in the extremities. The first signs of the disease, however, may be manifested by degenerative changes in the nervous system, rather than by a pronounced anæmia, and subacute combined degeneration of the cord results.

The earliest symptoms of this condition are the paræsthesias: numbness, tingling or a feeling of coldness in the extremities. As the name implies, this disease does not affect only one column in the spinal cord, but is "combined", and both dorsal columns and pyramidal tracts are involved.

The involvement of the pyramidal tracts is shown by an extensor plantar reflex or Babinski reaction, which is the earliest organic sign found in the disease, and by loss of the abdominal reflexes.

The involvement of the dorsal columns is shown by an early loss of sense of position, recognition of size, shape and form, and vibration sense. Too much stress cannot be laid on the importance of investigating these sensations in a suspected case of subacute combined degeneration of the cord. Sensations of pain and temperature are also affected, but usually at a rather later stage. An interesting point is that the knee jerks may be lost or exaggerated, according as to whether the dorsal columns or pyramidal tracts are earliest affected.

The gait is usually characteristic, being of the spastic-ataxia type.

Achlorhydria is an almost invariable finding in pernicious anæmia and subacute combined degeneration of the cord.

The essential feature of the blood picture is megalocytosis with a high colour index. The red cells are greatly reduced in numbers and exhibit great variations in size and shape. The leucocytes are reduced and a relative lymphocytosis is noted. The reticulocyte count is low and the blood platelets are diminished in numbers.

The treatment of pernicious anæmia is comparatively simple. Liver is the first essential, and should be administered in large doses. At least one and a half pounds of liver or its equivalent, as the extract, must be given daily to a pernicious anæmia patient when first seen. I think it is wise to give the patient half a pound of whole liver or kidneys daily and the remainder as extract. And it is important to note that all liver extracts have not the same potency.

The maintenance dose of liver which is given when the blood count reaches normal is half a pound daily.

In the treatment of subacute combined degeneration of the cord whole liver is more efficacious than liver extract, as certain properties of liver which are important for the cord lesion are lost in the extraction.

An abundant supply of fresh vegetables, fruit and red meat should be given.

It is not necessary to give hydrochloric acid as a routine procedure, but only under certain circumstances, that is, in cases of vomiting and dyspepsia or if diarrhœa is present.

The removal of septic foci should be undertaken when the blood count reaches normal.

Recently extracts of liver, which can be given intravenously or subcutaneously, have been introduced, and are valuable in cases of intractable vomiting in which liver extract cannot be given by mouth. As yet I have had only a limited experience of these new preparations, but they should prove of value in selected cases.

"Ventriculin" or extract of hog's stomach, in doses of 30 grammes daily, is also used by many people, but personally I have usually placed my faith in liver as its results are so good.

I have endeavoured to show that pernicious anæmia is nutritional in nature and due to the lack of a specific ferment in the stomach, which normally reacts with vitamin *B* and produces a substance essential for the development of the red blood cell.

Nutritional Anæmias Due to Diet Deficiency.

I shall now deal with the second group of nutritional anæmias—those due to dietary deficiency.

The first example is the nutritional anæmia of infants, which is present in nearly all babies to a certain degree, but may become very severe. This is due to the low mineral and iron content of milk (whether human or bovine), and the infant must depend for many months upon the store of iron which it derived from its mother before birth. The blood picture shows a diminished number of red cells of the small type with a low colour index.

The anæmia of scurvy is due to the deficiency of vitamin *C*, which is necessary for the development of the normoblast to the nucleated red cell. It is also microcytic in type and is readily cured by the administration of the defective vitamin.

Tropical nutritional anæmia follows the chronic malnutrition so prevalent in many of the overcrowded eastern countries. The essential deficiency has been shown to be vitamin *B* and the anæmia is of the megaloblastic type. It is readily cured by the administration of liver or yeast.

Simple achlorhydric anæmia is due to shortage of iron resulting partly from improper food and partly from defective absorption from the alimentary tract. It occurs most commonly in middle-aged women, and is greatly aggravated by pregnancy. Hydrochloric acid is invariably absent from the stomach,

and this is the chief predisposing factor to the disease, as iron is best absorbed in an acid medium. Furthermore, the dyspeptic symptoms which are so common in this condition cause the patients to adopt a diet consisting mainly of carbohydrates. The resulting anæmia is of the microcytic type with a low colour index. A characteristic feature is the presence of both lightly and darkly stained cells due to the variation in their hæmoglobin content, and an index of improvement is an increase in the number of the darker cells. The clinical features are weakness and pallor, a sore tongue and frequently inflammation of the fauces. The nails are usually harsh and brittle and atrophic in character.

The anæmia is readily cured by a diet containing sufficient quantities of meat, eggs and fresh vegetables, and the administration of large doses of iron, either *ferri et ammoniæ citras* in twenty gramme doses three times a day, or Blaud's pills in doses of twenty to thirty grains three times a day. Liver has no effect on this anæmia.

Von Jaksch's anæmia is found in infants under three years of age, and a large number of the cases occur in children who show evidence of rickets or some other type of nutritional disturbance, but some cases have been attributed to syphilis or tuberculosis. The symptoms are those of anæmia with marked weakness and disturbance of nutrition and of the gastro-intestinal tract. The red cells are very much reduced, the colour index being below one, but the striking feature is the marked increase in the leucocytes, which number as much as 30,000 to 40,000 per cubic millimetre. Myelocytes are usually present, and neutrophile cells predominate.

The treatment is essentially directed to the improvement of the general nutrition, the administration of iron, and exposure to sunlight.

Nutritional Anæmias Due to Defective Absorption.

The third group of nutritional anæmias is that due to defective absorption, and is usually associated with diseases of the intestines. Examples of this condition are tuberculosis of the bowel, with resulting stenosis, gastro-intestinal ulceration from chronic dysentery, infection with *Dibothriocephalus latus* and probably cœliac disease. The anæmia in these intestinal diseases may be megaloblastic, microcytic or even aplastic, according to the stage at which the development of the red cell is arrested.

Anæmias Due to Blood Destruction (Hæmolytic Anæmias).

An important representative of this type is acholuric family jaundice. This is characterized clinically by jaundice of varying degree from infancy and enlargement of the spleen. The fæces are dark and contain an excess of stercobilin, and the urine contains no bile pigments. The disease is hereditary and familial in type. Gallstones are a very common complication even in young subjects.

The blood picture is characterized by a striking increase in the reticulocytes, which rise to as high as 30%, and an increase in the fragility of the red cells. The anæmia is microcytic in type, that is, most of the red cells are small, anisocytosis is marked, and nucleated red cells are numerous. Nucleic acid is invariably increased in the urine. The treatment is splenectomy.

The combination of a high reticulocyte count, numerous normoblasts and increase of uric acid in the urine, which are evidences of active blood regeneration, is in vivid contrast to the blood picture and urinary condition found in the nutritional group.

Another example of hæmolytic anæmia is *icterus gravis neonatorum*. This probably results from an exaggeration of the normal physiological process of destruction of superfluous red cells after birth. Hæmorrhage may take place from the umbilicus, bowel or vagina or into the subcutaneous tissues. It usually commences on the third or fourth day, and the small sufferers from the disease usually died, until it was discovered that they could be cured by the injection of normal human serum in large quantities, ten to thirty cubic centimetres given intramuscularly, or better still, if practicable, intravenously.

Other examples of hæmolytic anæmias are the paroxysmal hæmoglobinuria of syphilis and blackwater fever. The predominating symptom in both of these conditions is the passage of very dark urine which is caused by excessive hæmolysis and resultant excretion of hæmoglobin in the urine.

The treatment of these conditions is blood transfusion, and the response is remarkable. I recently saw a man who was *in extremis* suffering from blackwater fever at the Sydney Hospital, under the care of Dr. Harold Ritchie, and his response to repeated blood transfusions was amazing. It is well to remember in this connexion that in the treatment of all hæmolytic anæmias liver, iron, arsenic, and the removal of septic foci are all of little value, but dramatic improvement usually follows blood transfusion.

There are several rarer forms of hæmolytic anæmia such as infections with the bacillus of gas gangrene, poisoning by aniline derivatives among workers in certain industries and acute hæmolytic anæmia. Their characteristics resemble closely those of the other types of hæmolytic anæmia, and I shall not weary you with a detailed description of them.

The Anæmias of Pregnancy.

I should like to say a few words about the anæmias of pregnancy. This subject has been the cause of a great deal of discussion and a considerable amount of confusion in recent years, and I think the reason is that any of the various types of anæmia which I have described may occur in pregnancy. First, there is the microcytic type of anæmia with a low colour index, due to iron deficiency. It can be readily understood that this type of anæmia will commonly occur in pregnancy in varying grades of severity. In pre-natal life the child must store sufficient iron for its first year of life and the liver of a newborn child contains five times the amount which is found in the liver of an adult. If the mother's blood is deficient in iron, or if her powers of absorption are poor, a severe grade of this type of anæmia will develop.

Secondly, there is the megalocytic type with a blood picture closely resembling that of pernicious anæmia. These occur very commonly in the tropics, and are probably associated with a diet deficient in vitamin *B* or with intestinal infection interfering

with its absorption. This type of anæmia occurs also in Australia during pregnancy, and is probably due to deficient storage or absorption of vitamin *B* or allied substances, and the ever-increasing demands of the growing fœtus.

Thirdly, the hæmolytic anæmias of pregnancy form a rare but very interesting group. The blood picture is typical of the hæmolytic anæmias in general. The red cells are greatly reduced in numbers, many normoblasts are present, and the reticulocytes are increased even to as much as 40% of the total red cells. The colour index is usually about one, the blood platelets are reduced in numbers and the fragility of the red cells is increased. The ætiology is associated with the acceleration of blood breakdown and repair which occurs in pregnancy, but the exact mechanism has not been fully worked out.

All these anæmias show their chief effects in the last three months of pregnancy and attention is usually called to the condition by the marked pallor of the patient. Slight icterus may be present and digestive disturbances such as vomiting and diarrhœa are common. Premature labour may occur.

The treatment varies according to the type. In the type due to iron deficiency large doses of iron and ammonium citrate, or Blaud's pills, are administered.

In the megalocytic type, liver should be given, and in the hæmolytic group blood transfusion is indicated. It is, however, frequently necessary to combine these methods of treatment, as the type of anæmia may not be pure.

The question of artificial termination of pregnancy may arise, but there is some risk attached to this procedure. The general consensus of opinion is that it is wiser to try to carry on the patient to full term by the use of the methods outlined, and to resort to artificial induction only if these fail.

Aplastic Anæmia.

Aplastic anæmia may be due to a variety of known causes: (i) Severe and overwhelming infections, (ii) poisoning by benzol, (iii) excessive exposure to X rays. Idiopathic or primary aplastic anæmia, however, constitutes a disease whose ætiology is unknown.

The main features of the disease are: (i) A severe inflammatory condition of the throat, (ii) an intense anæmia whose blood picture is characterized by the complete absence of all signs of activity on the part of the bone marrow.

No reticulocytes or normoblasts are found, and the red cells are of the mature type without any irregularities in size or form. The leucocytes are diminished in numbers, due chiefly to a loss of the granular forms which may represent only 5% to 10% of the total number. Eosinophile cells may be entirely absent, whereas the proportion of lymphocytes is usually about 80%. Immature leucocytes are not found, those present being well developed and mature. The blood platelets are greatly reduced in numbers and in consequence purpuric manifestations are common.

The prognosis is grave, and treatment is usually of little avail. Blood transfusion alone may prove beneficial.

It is sometimes difficult to distinguish aplastic anæmia from aleuchæmic leuchæmia in which there is found anæmia with a diminished number of leucocytes, a large proportion of which are lymphocytes. The distinction is made by the immature character of the lymphocytes in leuchæmia.

Agranulocytic angina, which has given rise to so much discussion in recent years, must also be differentiated. This disease is intensely interesting. It was originally described by Schultz nine years ago, and its essential clinical manifestations were stated to be "an acute onset with high fever, usually in a middle-aged woman, a course marked by ulceration of the throat and mouth, leucopenia characterized by a marked decrease or absence of granular leucocytes, a normal red cell and platelet count, and a fatal termination". Since this original description, much confusion has arisen, because the term agranulocytic angina has been transferred from its original meaning to a host of other conditions, particularly to those aplastic anæmias which are secondary to poisoning with arsenical and benzol preparations and applications of X rays.

An interesting point of discussion has been whether the intense infection of the throat destroys the leucocyte-forming portion of the bone marrow or whether the primary disorder is a depressed function of the bone marrow, and that infection of the throat is secondary.

Spirochætes, Gram-positive cocci of all types and Gram-negative bacilli are found in the throat, but it is believed by the majority of workers that these are secondary invaders and that the primary cause of the disease is lowered resistance and chronic ill health. A massive infection by the organisms mentioned above, then overwhelms the weakened bone marrow.

Three types of the disease are now described: (i) The fulminating type of Schultz with death in a few days. (ii) The type in which the illness is more prolonged and death occurs in a few weeks. (In these cases anæmia and purpuric manifestations may develop.) (iii) The relapsing type with cycles of attacks separated by months. The death rate is between 80% and 90%.

It should be remembered in the diagnosis that the onset of high fever in a middle-aged person with ulceration and membranous formation in the throat should, if the toxæmic symptoms are out of proportion to the extent of ulceration of the throat, lead to an investigation of the blood condition. Some cases have even occurred without marked throat infection, and the importance of making routine blood examinations in any case of long continued fever of unknown origin should be recognized.

The treatment is rather unsatisfactory. Blood transfusion is recommended, but the results are not striking. Vigorous local treatment of the throat should be undertaken, and more recently injections of leucocytic extract have been recommended.

Conclusion.

In conclusion I may say that I have not attempted to deal with various other diseases of the blood such as leuchæmias, but have endeavoured to keep to

the subject title of this lecture, "The Anæmias". My main object has been to show that modern work since the introduction of liver therapy has completely altered our whole idea of the ætiology and classification of these diseases. I hope that you will agree that these theories, although revolutionary, are well worthy of consideration.

Acknowledgement.

Much of the material for this paper has been derived from the Goulstonian Lectures delivered by L. J. Witts and published in *The Lancet* (March 5, 12, 19 and 26, 1932).

THE INFECTION OF THE PLACENTA AND OF THE FŒTUS BY INDUCTION OF LABOUR IN ECLAMPSIA.

By W. J. Penfold, M.B., Ch.M. (Edinburgh), D.P.H., B.Hyg. (Durham), M.R.C.S. (England), L.R.C.P. (London),

and

Hildred M. Butler, B.Sc. (Melbourne).

(From the Baker Institute of Medical Research, Alfred Hospital, Melbourne.)

This research was originally undertaken to investigate the origin of eclampsia. No satisfactory explanation of the eclamptic toxæmia has yet been offered, so that we felt justified in investigating afresh the possibility of its being of bacterial origin; and this in spite of the statement of Cruickshank, Hewitt and Couper[1] that there is no clinical, bacteriological or histological evidence to support the suggestion that the condition is due to the action of any animal or vegetable parasite.

The placenta has much to recommend it for the bacteriological investigation of this disease. It has been held by some authors[2] [3] to be the most likely source of the eclamptic toxin, so that it seemed likely that it would contain the organismal cause of the disease, if such existed. Moreover, it is available from every case.

Evidence has been brought forward recently showing that toxin production by certain organisms may be greatly favoured by a high carbon dioxide content of the medium.[4] [5] Further, *Brucella abortus* of Bang, which grows so profusely in the uterus, is greatly helped in its growth directly from the body by a high carbon dioxide content in the atmosphere to which its cultures are exposed.[6] The organisms grow profusely in the pregnant uterus and placenta of various animals, and it occurred to us that the high tension of carbon dioxide might be a helpful factor in determining such intrauterine growth.

It has been shown by Bell[7] and his co-workers (1928) that the pH of the blood of the fœtus is lower than that of the mother, and that a slight difference in the pH of the blood of the umbilical artery and vein exists to the extent of 0·13 of a pH unit. The lowering of the pH of the blood in traversing the fœtus is attributed by these workers to the large addition of lactic and carbonic acids made by the fœtus. According to Millet,[8] the fœtal tissues have also an extremely low pH, though as pregnancy advances, it approximates more closely to that of the maternal tissues. The pH of the placental tissue is also lower than that of the maternal tissues.

These considerations suggested to us that the use of a high carbon dioxide content of the atmosphere of the environment might promote the development of placental organisms, which had hitherto been missed, and, further, that a toxin might be obtained by the use of such an atmosphere that might be capable of producing convulsions similar to those found in eclampsia.

Bacteriological Technique.

The placenta was delivered in as careful a manner as possible and was placed immediately in a sterile metal container with a firmly-fitting lid and removed forthwith to the laboratory. The cultures were usually made from one-half to one hour after the delivery of the placenta. In no case was it longer than two hours.

On arrival at the laboratory the placenta was removed from the metal container and placed on a sterile towel. An area over one of the larger vessels was seared and blood was removed from the vessel with a Pasteur pipette and inoculated in approximately one cubic centimetre quantities into placental tryptic broth and into cooked meat medium. In some cases ordinary tryptic broth and McConkey's agar plates were also inoculated. A set of similar cultures was made from a second vessel. The placenta was then turned over and portion of the maternal surface thoroughly seared. Small portions of tissue were removed from the seared area and added to placental tryptic broth and cooked meat medium, two tubes being used in each case. Other areas of the placenta were similarly seared and portions of the tissue added to the media already inoculated. Thus, if organisms were present in one part of the placenta only, unless the infected area were very small, it would be unlikely that they would be missed, provided they could be cultivated in the media used.

One set of cultures was incubated in the air; the second set in an atmosphere of 10% carbon dioxide. As in the case of cultures from the vessels, ordinary tryptic broth and McConkey's plates were sometimes used. In no case was growth obtained in the last-named media when it had not developed in the placental tryptic broth and the cooked meat medium.

At the time of making the primary cultures, direct smears were made from the placental substance and stained by Jensen's modification of Gram's method. These preparations frequently revealed, sometimes in great numbers, the organisms isolated in our cultures.

As soon as growth appeared in the broth cultures, they were plated on blood agar and incubated aerobically and anaerobically and in an atmosphere of air *plus* 10% by volume of carbon dioxide. McIntosh's and Fildes's jars were used throughout for the anaerobic and carbon dioxide cultures. If Gram-negative bacilli were present in the broth cultures, they were also plated on McConkey's agar.

All broth cultures were incubated for one month before being discarded as showing no growth.

Many coliform organisms were obtained from these placentas and they were examined for cultural and biochemical properties. A number of anaerobic bacilli were isolated, which were not completely identified, except in the case of *Bacillus welchii* and *Bacillus tertius* strains.

Material.

In all, twenty-five placentas were examined, seventeen from cases of *ante partum* eclampsia, seven from *intra partum* cases and one from a *post partum* case. A *résumé* of this material, with the bacteriological findings, is given in Table I. As a control, placentas from ten cases of normal labour were examined. The technique employed was similar to that used with the placentas from cases of eclampsia. Seven of these placentas were apparently sterile, staphylococci were isolated from two, and a non-hæmolytic streptococcus from the remaining one. It seemed possible, in spite of our precautions in making cultures only from the substance as opposed to the surface of the placentas, that our cultural results might have been due to the occurrence, during delivery, of fæcal contamination of the surface. For this reason, six placentas from normal cases were smeared with emulsions of fæces by means of a swab and allowed to stand in the metal container for from one-half to one hour. They were then treated in the same way as placentas obtained from eclamptic patients. *Bacillus coli* was not isolated by our technique from any of these placentas.

All the organisms found in the placentas were tested for pathogenicity in rabbits and guinea-pigs. Special attention was directed to the power of the cultures to produce convulsions on inoculation into laboratory animals, and this power was only found in a significant degree with cultures of coliform bacilli. On inoculation into guinea-pigs and rabbits, broth cultures of these coliforms, grown in an atmosphere of 10% carbon dioxide, produced more marked convulsions than broth cultures of the same organisms grown in ordinary air.

These results reminded us of the claims of very early workers[9] [10] who also had been able to produce convulsions by means of cultures of organisms isolated from cases of eclampsia. An analysis, however, of the clinical material strongly suggested that the coliforms were introduced into the uterus by external interference after the eclampsia had already set in, so that they could not be the original cause of the disease. The external interference usually took the form of the induction of labour, which in every case was carried out by the introduction of a rectal tube into the uterus. When the *ante partum* cases, seventeen in number, are divided into two groups, those in which the labour was induced and those in which it was not induced, we find that in the seven cases (Numbers VII, X, XIV, XVII, XVIII, XX, XXIV) not requiring induction not one placenta showed infection with *Bacillus coli*. Of the ten placentas obtained from cases in which induction was practised, six (Numbers I, II, IX, XV, XVI, XIX) showed infection with coliform organisms, so that it is difficult to escape the conclusion that the induction of labour caused the infection. The eclampsia, however, existed without coliform infection of the placenta in all the non-induced *ante partum* cases and, therefore, in these cases at least, could not have been produced by such infection.

One placenta out of the seven derived from *intra partum* cases showed a coliform infection, and in this single case the labour was long and difficult from the presence of a contracted pelvis.

The placenta of the single *post partum* case was sterile within the limits of accuracy of our technique.

Our evidence, therefore, goes to show that the coliform infection of the placenta was in no way essential to the existence of the eclampsia, but was closely correlated with the induction of labour.

Grown either with or without carbon dioxide, the other organisms we found in the placentas did not reveal any definite capacity for the production of a convulsant poison. In eleven of the twenty-five placentas examined, non-hæmolytic streptococci were present. Two of these streptococci were strictly anaerobic. Seven of the eleven strains were found in placentas of cases in which labour had not been induced, while four came from cases in which induction had taken place. Apparently the external interference involved in the induction of labour did not have a great influence on the frequency of the occurrence in the placentas of non-hæmolytic streptococci. In the placentas derived from the women whose labours were normal, non-hæmolytic streptococci were discovered only once.

Staphylococci were isolated from five of the placentas derived from eclamptics. Their presence was associated with induction of labour in only one case.

Gram-positive bacilli were present in nine of the twenty-five placentas. Of these, two were toxogenic strains of *Bacillus welchii*, of which the cultures in animal test could be completely neutralized by serum. One other strain was of the same morphological type, but did not produce any significant amount of toxin. All three strains were obtained from the placentas of cases in which labour had been induced. Two of the remaining six Gram-positive bacilli were identified as *Bacillus tertius*. They were avirulent for guinea-pigs and rabbits and also unable to produce toxin. Both strains were recovered from patients in whom induction had been undertaken. The other four Gram-positive bacilli were not of well-recognized pathogenic types and did not come from placentas of cases in which induction of labour occurred. We do not feel warranted in discussing their possible origin. This evidence, therefore, is strongly suggestive that induction of labour is frequently associated with contamination of the placenta by fæcal organisms. We have obtained no evidence suggesting that any of the organisms we have found have a causal relationship to eclampsia.

The Relationship of Mortality in the Child to the Organisms Found in the Placenta.

Eight infants were either stillborn or died within four days of birth. The placentas associated with these infants were infected with *Bacillus coli* in six cases. Of the two infants associated with placentas not infected with *Bacillus coli*, one died from a fractured skull, while the other was premature, the fœtus being aged only thirty weeks. Of the other six infants of mothers with coliform infection of the placenta, five were delivered after induction of labour (Numbers I, IX, XV, XVI, XIX). In the case of the remaining infant the infection seemed to depend on a long and difficult labour associated with a contracted pelvis, but unassociated with induction. Of the five infants born of mothers in whom labour

had been induced and in whom also coliform infection of the placenta existed, two showed evidence of maceration, the degree of which was not, however, shown in the *post mortem* notes. It is unlikely that the deaths of these infants could have been due to the coliform placental infection if *Bacillus coli* had been first introduced during or after the introduction of the rectal tube to induce labour. Nevertheless, it is well known that *Bacillus coli* produces a toxin which rapidly causes necrosis of the skin. Whether this toxin plays any part in the maceration of the skin of the fœtus has not been investigated as far as we are aware. Of the other three infants, one which died four days after birth was found *post mortem* to have a bronchopneumonia, the bacteriology of which was not determined, while no *post mortem* examinations were made on the bodies of the other two infants. We are not in a position to say positively, therefore, what part *Bacillus coli* played in bringing about the deaths of these three infants, but in view of the well-known frequency of coliform infection of the new-born it is probable that it was at least a contributing factor to their deaths.

Consideration was given to the possibility that their deaths might have been due to the extra severity of the eclampsia in their cases. Three tests were used in investigating this point:

(1) Number of fits. It was found that the mothers suffering from *ante partum* eclampsia whose children did not survive, had on an average three and one-fifth fits before delivery, while mothers with *ante partum* eclampsia whose children did survive, had on an average three and one-third fits, that is to say, there was practically no difference in the number of fits as between the two classes.

(2) Amount of œdema. It is well known that œdema is associated with a favourable prognosis in eclampsia. On considering the average amount of œdema in the *ante partum* patients, we find that those whose children survived had less œdema than those whose children died. As far, then, as the œdema could be taken as an indicator, the eclampsia was more severe in the mothers of the children that did not die.

(3) Amount of albumin in the urine. The mothers suffering from *ante partum* eclampsia whose infants died, had on the average more albumin in the urine than the mothers of the surviving children.

In respect of these three tests, therefore, one suggests the mothers of the two groups of children were equally ill, one suggests that one group was more ill than the other, and the third test suggests the opposite, so that we have no reason to postulate that there was a substantial difference in the severity of the disease in the two groups.

It has been shown by the London Obstetrical Committee that the induction of labour in eclampsia is associated with a high fœtal mortality.[11] Bourne expresses surprise at this result of induction and suggests it is probably due to placental insufficiency or the long interval between the onset of the disease and the delivery of the child subjecting the infant to the action of maternal toxins for a longer time. We, on our part, believe that one, at least, of the dangers to the child of induction of labour in eclampsia is the contamination of the placenta and probably also the *liquor amnii* with coliform organisms, while possibly other intestinal organisms also may play a part. It seems likely enough that the diseases of the fœtus and of the new-born due to infection with *Bacillus coli* and other organisms may well take their origin from the placenta, or in the case of pneumonia from aspiration of a contaminated *liquor amnii*. In the case of neo-natal deaths, we attach little importance to infection through the umbilical stump.

Bacillus Coli Infections in the New-Born.

Ylppö[12] (1919) undertook blood culture in twenty premature infants who died between birth and the fifteenth day. He obtained twelve positive results, six of them being due to *Bacillus coli*.

There were seven placentas in our series, the fœtal vessels of which yielded blood containing coliform organisms. We regard these positive results as equivalent to positive blood cultures taken from the fœtus at the time of delivery. Only one of these seven babies survived.

Browne[13] (1921) has drawn attention to the frequency of coliform infections of the lung in the new-born, which he ascribes to the inhalation of infected material during or immediately after delivery.

Cruickshank[14] (1930) has drawn attention to the occurrence of septicæmias of the new-born accompanied by purulent pericarditis amongst other local lesions. Of twelve such cases of pericarditis, seven were bacteriologically examined; in three cases *Bacillus coli* was recovered. He also described forty-four cases of peritonitis in the new-born. In thirty-four of these there was a generalized septicæmia. From the whole forty-four both coliform organisms and streptococci were isolated.

Where in our cases the streptococcus was recovered from the blood of the fœtal vessels in the placenta, it was in every case associated with *Bacillus coli*. Where, on the other hand, streptococci were found in the placental tissue without *Bacillus coli*, then in no case were they recovered from the blood of the fœtal vessels of the placenta.

Cruickshank further described in the same report thirty-three cases of meningitis in eight hundred cases of neo-natal death. In twenty-three the bacteriology was fully investigated. In no less than eleven of the twenty-three *Bacillus coli* only was found. It was also found in some seven cases accompanied by streptococci. He reported, in addition, the occurrence of abscesses in various parts of the body of twenty newly-born children. No less than eight of these abscesses showed the presence of *Bacillus coli*.

Gibbens[15] (1932), in carrying out *post mortem* examinations on new-born infants, found three cases of meningitis due to *Bacillus coli*. In one case the organism was obtained by lumbar puncture half an hour before death. In another case with cerebral symptoms, but not a meningitis, the blood taken from the superior longitudinal sinus gave a pure culture of *Bacillus coli*. Two of the three cases of meningitis occurred in children where ample opportunity of

TABLE I.

Number of Case.	Pregnancy.	Induction of Labour.	Relationship of Fits to Labour.	Length of Labour (hours).	Fits. Total.	Fits. Before Admission.	Fits. After Admission.	Fits. Before Delivery.	Fits. After Delivery.	Oedema.	Maximum Albumin	Child.	Bacteriological Examination. Culture from Tissue of Placenta. Staphylococci.	Streptococci.	Gram-negative Bacilli.	Gram-positive Bacilli.	Culture from Blood of Placental Vessels. Staphylococci.	Streptococci.	Gram-negative Bacilli.	Gram-positive Bacilli.
I	1	+	Ante partum	15	6	1	5	6	0	+ + +	Solid.	Premature; macerated. Alive.		+	+	+				+
II	1	+	Ante partum	36	10	0	10	10	0	+ + +	+	Alive.	+	+	+	+		+	+	+
III	1		Intra partum	10	8	0	8	2	6	+ + +	+	Alive.								
IV	1		Intra partum		6	0	6	2	4	+ + +	+	Alive.								
V	4	+	Ante partum	3	1	1	0	1	0	—	+	Alive.								
V'	1	+	Ante partum	6	4	2	2	4	0	+ + +	+	Alive.					+			
VII	11		Ante partum		3	0	3	3	0	+ + +	Solid.	Alive.						+	+	+
VIII	1		Ante partum		6	0	6	4	2	—	+	Alive.		+				+	+	
IX	1	+	Intra partum	20	4	3	1	4	0	+ + +	+	Died after twenty-four hours. Stillborn.	+	+	+	+	+	+	+	+
X	1		Ante partum	23	3	0	3	3	0	+ + +	+	Alive.		+	+	+		+	+	+
XI	1		Ante partum		3	3	1	3	0	+ + +	+	Alive.	+	+		+				
XII	1		Intra partum	24	3	3	1	3	0	—	+	Alive.								
XIII	2		Ante partum	24	0	0	6	3	0	+ + +	+	Premature; stillborn. Alive.								
XIV	1	+	Ante partum	13	1	1	0	1	0	+ + +	Solid.	Alive.		+						
XV	6	+	Ante partum	44	1	0	1	1	0	+ + +	+	Alive.		+						
XVI	1	+	Ante partum	24	3	0	3	3	0	+ + +	+	Premature; lived four days. Stillborn; macerated. Alive.								
XVII	1		Ante partum	22	5	1	4	5	0	—	Solid.	Alive.		+	+			+	+	
XVIII	1		Ante partum	?	6	2	4	6	0	+ + +	+	Alive.								
XIX	1	+	Ante partum	15	2	1	1	2	0	+ + +	+	Lived one hour. Alive.		+		+		+	+	+
XX	1		Ante partum	? 24	3	2	1	3	0	—	+	Alive.	+			+	+			
XXI	1		Intra partum	? 24	6	3	3	3	3	—	+	?	+	+		+				
XXII	1		Post partum	24	5	0	5	5	5	+ + +	+	Alive.								
XXIII	1		? Intra partum	3 days	3	0	3	3	0	+ + +	+ +	Stillborn; fractured skull.		+						
XXIV	1	+	Ante partum	13	2	0	2	2	0	+ + +	+ + +	Alive.								+
XXV	1		Ante partum	25	1	0	1	1	0	+ +	+	Alive.								

infection of the maternal uterus was possible, from induction in one case and a difficult labour requiring forceps in the second case.

There is a disinclination on the part of some authors, for example Cruickshank (1930),[14] to attribute a pathogenic rôle to *Bacillus coli*. It is difficult to understand this in view of the various studies that have been made on the virulence and toxogenicity of the organism and the clinical conditions in which it is the only bacterium present. Vincent[16] and other workers have shown that *Bacillus coli* is able to produce neurotropic and enterotropic toxins. We have personally been able to confirm these results, but we have found both the cultures and filtrates of some strains able to produce marked convulsions in the guinea-pig. This Vincent apparently did not observe.

In addition, *Bacillus coli* cultures produce skin necrosis in rabbits and guinea-pigs, which has been very extensive with some of our strains.

In testing the virulence of our coliform strains by intravenous administration in rabbits, we frequently found, in the case of those animals that died early (within ten to twelve hours after administration), that few, if any, macroscopic changes were found *post mortem* in the body. This should be kept in mind when investigating the cause of death in the new-born. Fatal *Bacillus coli* infections may be present without gross macroscopic lesions.

In view, therefore, of the frequency of the contamination of the placenta with *Bacillus coli* in induced labour and the various infections of the new-born in which *Bacillus coli* is present, either alone or accompanied by some other organism, it is difficult for us to escape the conclusion that the source of the infection in many of these cases is the placenta, and that these infections constitute an important danger to the fœtus from the induction of labour. We believe that the infection of the placenta with *Bacillus coli* and other organisms is effected by carrying out the operation of induction of labour, or, alternatively, it occurs during the long interval which elapses between the insertion of the rectal tube and delivery. The liability to contamination in this latter case would be greater the longer the interval.

If our ten *ante partum* cases in which induction of labour was practised be divided into two groups, namely, those in which the placenta was infected with *Bacillus coli* and those in which it was not so infected, it is found that the interval between the insertion of the rectal tube and the delivery of the child was on the average longer in the case of the infected than in the non-infected in the proportion of forty-one to thirty-one hours approximately.

In this connexion we should like to recall the particulars of a case of pre-eclamptic toxæmia which required induction of labour. The placenta in the case was infected with *Bacillus coli*. The patient had a febrile puerperium and on the tenth day the uterus was swabbed for bacteriological examination. *Bacillus coli* and a diphtheroid were recovered. The fact that *Bacillus coli* was found in the uterus speaks against the *Bacillus coli* of the placenta being a casual contamination occurring in delivery.

It seems likely that the risks from intrauterine sepsis are even greater for the child than for the mother. Aspiration of contaminated *liquor amnii* into the air passages may well account for some of the pneumonias of the new-born, but we have made no bacteriological examinations of the amniotic fluid.

The rapid fall in infantile mortality which has occurred in recent years has not affected the mortality due to stillbirths or neo-natal disease. We trust that the risk to the infant of intrauterine manipulations and consequent bacterial infection may be greatly lessened through a consideration of the results recorded in our paper.

Summary.

1. We have not been able to isolate from the placentas of eclamptics any organism which appears to us to be ætiologically related to eclampsia.

2. Coliform organisms were frequently found in the placentas of a series of women suffering from eclampsia.

3. The presence of these organisms was closely correlated with the induction of labour.

4. Their presence was likewise closely associated with a high fœtal or neo-natal mortality, but we have no positive evidence from our series that this mortality was caused by the placental infections.

5. The frequency with which coliform infections of the new-born occur, described in the literature, has probably been largely due to infection of the fœtus from the contaminated placenta.

6. It is evident that the present technique employed in the induction of labour is not beyond criticism.

Acknowledgements.

Our best thanks are due to the medical staffs of the Women's and the Queen Victoria Hospitals, Melbourne, for their continued interest in this work, for the supply of placentas, and for access to the clinical notes which have been of the greatest help to us.

We regret that for various reasons it was not possible to obtain sufficient *post mortem* information relative to the dead infants.

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PLUMBING.

By H. C. RUTHERFORD DARLING, M.D., M.S. (London), F.R.C.S. (England) *et cetera*,

Honorary Surgeon, The Coast Hospital, The New South Wales Community Hospital, and Lidcombe State Hospital, Sydney.

A DISTINGUISHED but facetious physician once remarked that the primitive, rudimentary and unromantic science of plumbing boasted of a "super-science" or "higher plumbing" which formed a cult widely and universally followed and designated the science of surgery. The weapons employed in both sciences may vie with each other in complexity and are innumerable, but whilst the plumber is well content to pursue his avocation with the tools used by generations of his forbears, no youthful surgeon is happy until he has devised some new and cunning variation of some implement of his art, whereby he may both win renown and possibly more successfully ply that art.

Metaphorically speaking, however, a huge abyss lies between the indications for the use of these various implements in the two cases. For whereas the plumber's spanner varies directly in hugeness with the size and delicacy of the nut he is unscrewing, the instrument of the surgeon varies in the inverse direction, so that we see him approaching that robust organ, the brain, with a powerful electrically-driven drill and the fury of his efforts to uncover that unhappy structure would lead one to believe that he regards this, as he does most other organs, as vestigial. Both sciences may be said to find their highest development in the cunning manipulation of pipes and tubes, which are cleared, stopped, patched, and short-circuited at will, and only a surgeon would really appreciate the savage glee that fills the breast of the triumphant plumber who has, after some labour and more emotion, pierced the obstruction in a refractory drain.

As a devotee to "higher plumbing" one learns that the majority of "leaks" in the human frame follow upon disease or operations on the pelvic organs.

From an anatomical standpoint they may be grouped into "sinuses" and "fistulæ".

A sinus usually arises through an intraperitoneal abscess penetrating the abdominal wall and discharging externally.

If anything such as a foreign body or a portion of dead tissue prevents healing, a tubular ulcer or sinus results. If the discharge from a sinus slowly increases in quantity, the pus should be examined for tubercle bacilli and the clubbed hyphæ of *Streptothrix actinomyces*.

A fistula is an abnormal communication between the skin and bowel or other hollow viscus. In other words, it may be defined as a tubular ulcer, one extremity of which opens into a canal lined by mucous membrane, whilst the other penetrates the skin. It may be congenital or acquired through injury, operation, gangrene, suppuration, tuberculous or malignant disease.

The differentiation between a sinus and a fistula may often be difficult, and require careful clinical as well as radiological examination.

The passage of urine, fæces or flatus through the track, however, establishes the diagnosis of fistula.

Hypogastric Sinuses.

In the early part of the present century most intractable sinuses were frequently met with following operations for the removal of infected uterine appendages. Like other cutaneous sinuses of that period their source was most commonly due to some foreign body, such as one or more infected ligatures or pieces of indiarubber (patches) from a glove.

In the series under review hypogastric sinuses arose from (*a*) retrocaecal actinomycosis, (*b*) self-inflicted injury, and (*c*) post-operative suppuration of an intramural uterine hæmatoma.

Retrocaecal Actinomycosis.

A *multipara*, aged forty-six, came for examination on April 14, 1929, on account of two sinuses, one in the right iliac region and the other in the median plane of the hypogastric zone, that had been present for six months and five weeks respectively.

Three years previously she had been admitted into a metropolitan hospital on account of abdominal distension, vomiting and colicky pains of three days' duration; but, after examination, was discharged with the injunction that the trouble could be overcome by the administration of suitable aperients. Subsequently an indefinite tender mass was located in the right iliac fossa, but radiological examination failed to disclose any abnormality of the colon.

Four months later, as the mass was increasing in size, exploratory laparotomy was suggested with the idea of determining the nature of the morbid swelling. A thickened *caput caeci*, together with the ampulla of the right uterine tube and ovary and a retrocaecal appendix were found to be bound up in dense adhesions to form a mass occupying the right iliac fossa. Further radiological examination failed to disclose any abnormality of the gastro-intestinal tract. The appendix was not visualized.

Two years later, as the mass had increased to the size of a coconut, a further operation was performed when the terminal part of the ileum was implanted into the transverse colon. For the last month the patient had been complaining of marked frequency of micturition and strangury.

On examination on April 14, 1929, a large irregular tender fixed mass, the size of a seven months pregnant uterus, could be felt arising out of the pelvis as high as the subcostal plane and filling up the right iliac fossa and both umbilical and hypogastric zones.

The iliac sinus lay 2·5 centimetres (one inch) below the centre of a McBurney's incision, whilst the other was found 6·25 centimetres (two and a half inches) below the umbilicus; both emitted a profuse purulent discharge.

Microscopical examination of the pus revealed Gram-negative cocci and a few fungal hyphæ which were too scanty to be identified. A further examination revealed numerous pale sulphur granules which in Gram films showed much mycelium, and in fresh preparations the typical clubbed hyphæ of *Discomyces bovis* (*Streptothrix actinomyces*).

The urine was acid in reaction, had a specific gravity of 1025, and presented traces of blood and acetone. On allowing the test tube to stand subsequent to boiling the depth of deposit was assessed as "almost solid".

Tincture of iodine (French Codex) 0·6 mil (ten minims) was administered three times a day, and the dose gradually increased by 0·18 mil (three minims) daily until a maximum dose of two cubic centimetres (half a drachm) three times a day was reached.

Two months later the sinus in the right iliac fossa had healed and only a small amount of thin seropurulent discharge exuded from the hypogastric sinus. The inflammatory mass was reduced to a swelling the size of a cricket ball in the right iliac fossa. Pathological examination of the discharge on September 14, 1929, showed numerous pus cells; there was no evidence of *Streptothrix actinomyces*, but diphtheroid bacilli grew on culture.

An autogenous vaccine, standardized to 1,000 millions per cubic centimetre, was prepared, but after the third weekly

injection of 0·25 cubic centimetre the patient refused further treatment and was temporarily lost sight of for two and a half years.

I am indebted to the medical superintendent of a metropolitan hospital for the following notes.

The abovementioned patient was admitted to this hospital on March 9, 1931. She complained that since a gastro-enterostomy was performed, at another hospital two years previously, she had had a discharging sinus at the umbilicus. She had also noticed a lump in the left breast for two months.

On examination there was a sinus at the umbilicus with a purulent sanious discharge. A large soft mass was palpable in the left breast, the mass was attached to the skin and freely movable, skin had the appearance of *peau d'orange*. The umbilical fistula was injected with bismuth paste, and a catheter introduced. By X ray examination the catheter was shown to pass downwards and backwards to the level of the symphysis, it was then curved on itself to the right, and above this. On March 25, 1931, a local amputation of the left breast was performed. The pathological report on microscopic examination was scirrhous carcinoma. On April 28, 1931, radical removal of the left breast was performed together with removal of glands of axilla. On March 25, 1931, the umbilical fistula was explored and the track of sinus opened up. The patient was discharged on June 11, 1931, with the wound healed.

The patient, who was in moderate health, presented herself to me for re-examination on September 16, 1932. The hypogastric sinus and umbilicus had been excised and through the cicatrix an incisional (ventral) hernia, the size of a large orange, had developed, whose neck readily admitted three fingers.

A large irregular tender fixed mass, the size of a coconut, could be felt lying deeply in the right iliac fossa.

Sinus Artefactus.[1]

A young unmarried woman of twenty-five was referred on May 26, 1932, on account of two sinuses, one in the left iliac fossa and the other in the hypogastric zone, which had followed right salpingo-oophorectomy for salpingitis and small right ovarian cyst performed at a district hospital on July 10, 1928.

The abdominal sinus had been excised on five separate occasions, following which various plastic procedures had been performed. The Wassermann test gave no reaction, and radiological examination failed to reveal any opaque foreign body.

On February 3, 1929, the excised sinus was submitted to the Department of Pathology, and the report read: "Chronic sinus of abdominal wall appears to be tuberculous as there are several well-defined giant cell systems embedded in a mass of dense fibrosis."

In December, 1930, the discharge from the sinus was examined pathologically, but no tubercle bacilli were detected and the guinea-pig inoculation test was without result. Menstruation had been in abeyance since July, 1927.

The patient had frequently sought advice and treatment at various other public hospitals.

Appendicectomy was performed at a country hospital in 1923. Partial oophorectomy, separation of adhesions and internal shortening were carried out at a metropolitan hospital on July 14, 1927, for retroversion of uterus, left ovarian cyst and peritoneal adhesions. Radium treatment had been used at a metropolitan hospital on July 12, 1929. Blood transfusion had been given for secondary anæmia complicating an abdominal sinus and pyelitis at a metropolitan hospital on November 5, 1930. She was discharged on January 9, 1931, with the anæmia relieved, sinus unrelieved, and the pyelitis cured.

On examination the lower part of the abdomen was seen to bear the imprints of much surgical intervention. Amongst the median infraumbilical cicatrices and lying one and a half inches above the pubis was a sinus admitting a Number 4 Hegar dilator and running obliquely downwards behind the symphysis for one and three-quarter inches. Surrounding the sinus was a tender indurated area the size of a hen's egg.

In the left iliac region, at the bottom of a deep fold produced by an adherent scar, was found a further opening which communicated by means of an oblique subcutaneous track with the main sinus.

[1] This patient was shown at a clinical meeting of the staff of the New South Wales Community Hospital on September 1, 1932.

The discharge from both openings was profuse and hæmorrhagic, and the patient volunteered the statement that six rolls of cotton wool were used weekly to absorb the discharge.

An area of induration lay between the pubis and the body of the uterus which was anteverted and anteflexed.

The radiological report, dated June 21, 1932, read as follows:

X ray examination of pelvis subsequent to lipiodol injection of the sinus and uterus.

Pelvis. I cannot detect any involvement of the bone.

Uterus. The left tube is patent. The right tube is apparently absent. There is no connexion between the uterus and the sinus on the anterior abdominal wall. The sinus on the anterior abdominal wall extends down towards the inner aspect of the symphysis pubis.

On July 4 the patient had a profuse attack of hæmaturia. Pathological examination of the urine revealed numerous red blood cells, but no pus cells or casts. No growth occurred on culture, nor were any tubercle bacilli detected.

When she was seen on July 21 the calibre of the sinus was considerably reduced, but the inferior margin of the orifice presented a peculiar mouse-eaten appearance and recent blood was exuding therefrom.

The patient was admitted to the New South Wales Community Hospital for observation on August 15, and at a Medical Board held on the following day the following conclusions were arrived at: (*a*) The condition was one of *sinus artefactus*. (*b*) The injury was produced by either the nail of the little finger or a safety-pin. (*c*) Both arms should be put up in plaster of Paris, the forearms crossed so that the hand rested on the opposite shoulder.

The dermatologist to the Hospital, Dr. J. Whitton Flynn, drew attention to a syndrome frequently present in association with *dermatitis artefacta* comprising anæsthesia of the conjunctivæ and palate, Magdalene countenance, fine tremor of the fingers and patchy anæsthesia of the skin; all of which were present in the case under discussion.

In the light of subsequent events one might question the applicability of any reformation.

The evening report on August 17 read as follows:

4 p.m. general anæsthetic, arms put up in plaster; 4.35 p.m. patient returned from the theatre; 6.30 p.m. two arms and one hand in plaster; 7 p.m. two arms in plaster, both hands free; 7.30 p.m. one and a half arms in plaster; 7.35 p.m. one arm in plaster; 7.45 p.m. half an arm in plaster; 8 p.m. arms and hands free of plaster; 9 p.m. bright red blood-stained discharge from both sinuses.

Under threats of dismissal the patient consented to be put up again in plaster provided her arms were folded and hands left free. This operation was carried out on August 19, and twenty-four hours later the discharge from the sinuses had practically ceased.

A bright hæmorrhagic discharge reappeared on August 23, so both hands were bandaged and a strong binder was applied from breast to knee.

On August 28, as the sister-in-charge had reported the wounds healed, the patient requested to be allowed to go home. On examination it was found that the abdominal binder had been pulled up and an actively bleeding suprapubic sinus, half an inch deep, was present.

The patient's hands were therefore encased in boxing gloves and the latter firmly secured to the plaster casing by adhesive strapping.

By September 3 both sinuses had healed and the patient was allowed to get up.

On the nurse's attention being diverted the patient escaped to the lavatory and subsequently a small ulcer, the size of a pea and a quarter of an inch deep, discharging blood-stained serum, was discovered.

The base of the ulcer presented the peculiar mouse-eaten appearance already recorded.

The patient did not deny that the injury had been produced by a safety-pin.

On September 6, as both sinuses were again soundly healed, the boxing gloves and plaster casing were removed and the patient was discharged.

Post-Operative Suppuration of an Intramural Uterine Hæmatoma.

An unmarried woman of thirty-eight sought advice on account of multiple fibroids filling the pelvis and rising well above the

brim, so that they could be readily palpated in the lower part of the abdomen. *Per vaginam* the cervix appeared to be attached at the central portion of the mass and the fundus could not be felt.

The patient refused to submit to hysterectomy as she had received a proposal of marriage. Fourteen subserous and interstitial myomata were removed through twelve separate incisions, leaving a very ragged-looking uterus. A flap of peritoneum was drawn over the fundus so as to prevent the formation of adhesions and to avoid the small intestine becoming adherent to the uterine incisions.

The convalescence was uneventful until the eleventh day when, twenty-four hours after removal of the stitches, the temperature suddenly rose to 39·4° C. (103° F.). On the fifteenth day sinus forceps were inserted into the wound and about six ounces of pus were evacuated. An intractable suprapubic sinus formed which resisted all efforts to secure healing for two and a half months.

On March 6 a slough in the centre of which was a thin sheet of fibrous tissue, about the size of half a crown, evidently part of the capsule of one of the tumours, was discharged from the sinus. By March 27 the sinus was soundly healed.

A dead space had evidently been left in one of the cavities produced by the enucleation and the resulting hæmatoma had become infected.

Hypogastric Fistulæ.

Three types of fistulæ may be met with in the hypogastric region: suprapubic vesical fistula, hystero-cutaneous fistula, and stercoral fistula.

Suprapubic Vesical Fistula.

A persistent vesical fistula is of relatively common occurrence and has been estimated to follow 1·2% to 2% of suprapubic operations performed for simple enlargement of the prostate gland. These patients are in a very much worse plight than if they had refused operation and resorted to catheter life.

The fistula is commonly associated with incomplete removal of adenomata, a valvular fold or fibrous contraction at the internal meatus or in the prostatic bed, a large diverticulum, or non-absorbable suture material. In a large percentage of these cases it is difficult or impossible to pass a catheter into the cavity of the bladder owing to the tip of the instrument hitching against a diaphragm roofing over the remains of the prostatic cavity. Over a quarter of a century ago Sir John Thomson-Walker pointed out that post-operative urethral obstruction was prone to occur from the formation of scar tissue at the internal meatus where the vesical mucosa was torn across, or at the membranous urethra where the urethra was severed.

Amongst the rarer causes of fistula are adhesions of the cystotomy wound to the abdominal scar or pedunculated nodules or flaps of tissue obstructing the vesical outlet.

The orifice of the fistula is usually small and may be surrounded by a collar of granulation tissue or placed at the bottom of a depressed scar.

If the urine be heavily infected and constant cleanliness be not observed the scar becomes thick and red, and the skin irritated and excoriated.

Commencing obstruction at the vesical neck may be suspected after prostatectomy when the onset of urethral micturition is delayed or increasing difficulty is presented in passing a curved catheter for the purpose of washing out the bladder.

In passing a catheter it must be remembered first that the vesical orifice is not in alignment with the direction of the urethra, and second that there is absence of guidance of the prostatic urethra; hence it is often difficult to hit off the vesico-prostatic opening, frequently contracted, with the point of the instrument.

In cases in which vesical lavage is not employed Mr. K. M. Walker recommends passing a full-sized Lister's bougie fourteen days subsequent to the date of the operation. If narrowing be noted, bougies should be passed at regular intervals until it be proved that no recurrence is taking place.

If the suprapubic track has not healed by the end of five or, at the latest, six weeks, a series of Lister's metal bougies of increasing size are passed and a large Number 16E or 18E Jacques catheter is tied in the urethra for seven to ten days, during which period the bladder is irrigated twice or thrice daily.

If difficulty be encountered in carrying out this manœuvre, the index finger may be inserted through the suprapubic opening to meet the tip of the bougie, which is pushed through the dimple or button of granulation tissue at the vesical neck and dilatation is proceeded with.

On the other hand, Mr. J. Swift Joly recommends dissecting out the fistula and adequately dilating the vesico-prostatic opening with the tip of the finger. An indwelling catheter is then placed in the urethra and the bladder closed.

If the above methods of treatment prove ineffectual, or if the suprapubic track has not healed at the end of eight weeks, the fistula should be dissected out, the bladder freed from adhesions and completely mobilized, the patient placed in the Trendelenburg position and the prostato-vesical opening carefully examined.

The granulation tissue at the vesical neck has now become converted into dense fibrous tissue resembling a traumatic stricture and is unlikely to yield to mere dilatation.

The fibrous ring should be divided posteriorly with long curved scissors and then removed from each side of the vesico-prostatic opening, the prostatic cavity being thus thrown freely open into that of the bladder. Any pedunculated adenomatous nodules or flaps of prostatic tissue are dissected from the prostatic bed. In the majority of cases the scar tissue can be dissected out, a Macalpine's combined drainage and irrigation tube is then inserted and continuous irrigation of the bladder with a dilute solution of oxycyanide of mercury commenced.

The prevalent practice of passing a 12/15 steel bougie through the urethra every week for months on end is to be condemned.

When I was appointed to the honorary staff of the State Hospital some five years ago twenty-two patients with persistent vesical fistulæ, all referred from metropolitan hospitals, were to be found congregated in the genito-urinary ward of that institution.

These cases were classified as follows:

(*a*) Contracted prostatic cavity complicated by severe genito-urinary sepsis	16
(*b*) Calculus impacted in prostatic bed	2
(*c*) Non-removal or incomplete removal of prostatic adenomata	2
(*d*) Malignant disease of the prostate	1
(*e*) Large infected diverticulum	1

Cardiac degeneration, hæmoglobin value and failure to pass the customary renal efficiency tests precluded operation in all but two cases which ranked as borderland or doubtful operation risks. These patients requested operation; in one, a calculus impacted in the neck of the bladder was removed, but the patient died on the fifth day; the other, in which the great bulk of an adenoma had been left behind, recovered.

As surgeons are so fully cognizant of the harmful effects produced upon the kidneys by pre-operative prostatic obstruction and universally apply tests to assess the function of those organs, why do they fail to appreciate the baneful results of post-operative vesical obstruction where the two lethal B's—block and bacteria—work in conjunction?

On rare occasions suprapubic fistulæ may be associated with perineal prostatectomy.

A "transfer" from a metropolitan hospital arrived at the State Hospital on July 7, 1931, bearing a note, which read as follows:

> Patient had a stone removed from bladder sixteen weeks ago and about six weeks later perineal prostatectomy was performed. Patient later developed a fistula between bladder and rectum and a colostomy was done to give it a chance of healing. This, however, did not succeed and patient was considered unfit for any further operative treatment.

The patient, aged 61 years, whose general health was considerably impaired, presented three recent operation scars: left iliac (Littré's) colostomy, and transverse suprapubic and transverse (semilunar) perineal incisions.

The pelvic colon had been opened at the apex of the loop, but no attempt had been made to arrest the downward passage of peristalsis by severing the loop at the level of the skin.

Both perineal and suprapubic incisions were interrupted in the median plane by fistulæ; that on the perineum discharging urine and pus, whilst the suprapubic opening transmitted a de Pezzer catheter for the purpose of draining the bladder.

The urine was offensive and ammoniacal, had a specific gravity of 1010, and contained a large amount of albumin and traces of blood. There was a heavy deposit of thick ropy pus which five or six times a day blocked the de Pezzer catheter leading to violent straining and severe pain, which persisted until relief was obtained through the contained urine escaping *vid* the perineal and colostomy openings.

With considerable difficulty a 6/9 Lister's bougie could be passed into the bladder, whose capacity was estimated to be between three and four ounces. The injection of 0·2 gramme of indigocarmine into the buttock failed to colour the urine in forty-five minutes. The patient was placed on urinary antiseptics and the de Pezzer catheter replaced by a Macalpine's combined drainage and irrigation tube.

Continuous irrigation of the bladder with weak oxycyanide of mercury solution was maintained for three weeks, during which time the urethra was intermittently dilated up to full size.

When the patient was seen on August 26, although the perineal sinus was still freely discharging pus, there had been no escape of urine for fourteen days. The capacity of the bladder was four ounces, and there had been no escape of urine from the colostomy opening or *per anum* for four days. The Macalpine's tube was removed, an indwelling catheter tied in the urethra, and the bladder irrigated twice daily.

By September 9 the patient was feeling considerably improved in health, the urine was less foul but still presented a heavy deposit of pus.

The radiological report dated October 10, 1931, read as follows:

> A number of small areas of calcification in bladder area about the size of seven peas. There are several similar areas at the lower end of the left ureter. These shadows are probably due to vesical calculi and perhaps calculi in the lower end of the left ureter.

On October 15 the patient passed a small pea-like stone *per anum*, and four days later a similar calculus was discharged through the distal colostomy opening.

On October 16 the patient had two rigors and subsequently complained of loss of appetite and energy, thirst and frequent headaches. The urea concentration test revealed the concentrating power of the kidneys to be 0·9%. On October 28 intermittent vomiting commenced and the patient died from uræmia two days later.

Stercoral Fistula.[1]

Human beings whose intestinal contents are escaping through an artificial opening upon the surface of the body, are very unhappy individuals, and will willingly submit to severe and dangerous operations to be free of their trouble.

An artificial anus is an opening from the intestine to the exterior of the body which is so arranged that all the contents of the intestine pass out of the opening and none go beyond. On the other hand a stercoral fistula is defined as an abnormal track leading from the lumen of the bowel to the surface and discharging only a portion of the intestinal contents. Such a fistula usually originates either from disease of the intestine or from injury sustained by the bowel during the course of operations, especially those performed for chronic suppurative diseases of the tubes and ovaries in which dense intestinal adhesions are present.

If the involved loop should become adherent to the parietal peritoneum prior to the formation of the fistula, the channel of communication will be short and fusion of skin and mucous membrane tends to occur at the mouth. On the other hand, if an abscess develops in relation to the damaged bowel and subsequently tracks to the surface, the fistula will be of considerable length and lined by granulation tissue secreting a variable quantity of pus (pyostercoral fistula).

Of somewhat similar character are the long tortuous sinuses travelling between coils of intestine, such as may pass from the surface deeply into the pelvis in cases of tubal or ovarian suppuration, in which the intestine has been implicated.

So long as the distal gut remains unobstructed, the opening in the bowel is usually a comparatively small one, allowing only a small portion of the intestinal contents to escape, and exhibiting a distinct tendency to close spontaneously. Spontaneous closure, however, seldom occurs if the fistula has become lined by skin or mucous membrane.

Three types of operation may be undertaken for the cure of a pyostercoral fistula. The abdomen should be opened, the intestinal end of the track sought for and divided and the opening in the bowel closed. The remnants of the fistula are either excised or the divided end is inverted and the track left to heal by granulation.

When the opening is a large one and closure by invagination would unduly narrow the bowel lumen, it may be necessary to resect the fistula together with the involved segment of intestine.

Long tortuous fistulæ, traversing coils of intestine, generally require one of the forms of intestinal occlusion such as ileo-sigmoidostomy (unilateral complete exclusion). In such cases exposure of the opening in the bowel is difficult and entails great risk of diffusion of infection; while, when exposed, the

[1] This portion of the paper was read at a clinical meeting of the staff of the New South Wales Community Hospital on May 5, 1932.

opening may be in a part of the bowel so fixed by adhesions to surrounding parts and so deeply placed that suture is impracticable.

On the other hand, certain patients in whom ileo-sigmoidostomy has been performed have subsequently suffered either from accumulation of gas in the proximal part of the colon or from reversed peristalsis causing the excluded segment to be kept constantly full of fæcal matter, thereby necessitating secondary colectomy. Attempts have been made to overcome this difficulty by anastomosing the caecum and sigmoid colon (caeco-sigmoidostomy of Giardano-Bergmann), but this procedure tends to create an endless colon in which the fæces move in a circle. On the other hand, Ochsner divides the colon proximal to the anastomosis and brings the upper end out on the left flank so as to secure permanent drainage.

The stump of the ileum may advisedly be brought out on the opposite flank so that the excluded segment can be irrigated.

The following case of pyo-stercoral fistula has recently been under my care at the New South Wales Community Hospital.

A *multipara*, aged fifty-one years, came for examination on February 19, 1932, with a history of double salpingo-oophorectomy performed at a metropolitan hospital in 1923, since which date there had been a persistent discharge of intestinal contents through the operation wound.

Three months prior to examination, a smart hæmorrhage had taken place when about a pint of blood was lost *per rectum* and through the fistula. The action of the bowels had been kept regular by taking a dessertspoonful of paraffin thrice daily.

On examination an irregular broad, red, inflamed, depressed, median scar was seen, extending from the umbilicus to just above the pubis, the maximum width of which was 7·5 centimetres (three inches). In the centre of the cicatrix were two sinuses about the calibre of lead pencils discharging pus and fæcal material. The scar was adherent to the deeper structures, and an indefinite mass could be felt deep to the left iliac region to which it appeared to be attached.

There was no occult blood in the stools. The Wassermann test on the blood gave no reaction, and urinalysis revealed no abnormality.

The radiological report, dated February 18, 1932, read as follows:

> X ray examination was made after injection of the fistula with lipiodol, and the bowel with barium. The fistula leads almost straight downwards for three and a half inches, and connects with the sigmoid colon. The sigmoid colon is irregular and constricted, but there was no delay in the passage of the barium enema.

At operation, on February 29, 1932, a circular incision was made around the fistulous track, the margins were dissected up and sutured over the orifices, so that there could be no leakage during the operation. The remainder of the cicatrix was excised, the individual layers of the abdominal wall were defined, and the peritoneal cavity was opened.

The fistula communicated in the left iliac fossa with a mass, the size of a closed fist, composed of matted, small intestine and securely fixed to the upper sigmoid colon. The appendix, densely adherent to the posterior abdominal wall, descended into the pelvis through an artificial diaphragm composed of matted coils of small intestine and omentum forming a barrier between true and false pelvis. Direct attack on the fistula was precluded owing to the extensive adhesions present.

After prolonged search a short loop of the lower sigmoid was drawn up through a hole torn in the adherent omentum. The ileum was then divided 12·5 centimetres (five inches) from the caecum, its distal end was invaginated and closed over with a purse-string suture, and its proximal end was reimplanted into the lower end of the sigmoid by end to side anastomosis. Temporary valvular caecostomy was performed, Senn's method being used, and a de Pezzer catheter was inserted into the caecum. The abdominal wound was stitched up in layers, the fistula being implanted in its lower angle.

The customary after-treatment recommended for enterectomy was carried out. A smooth convalescence was marred by the development of extremely obstinate constipation, which led to profuse discharge of intestinal contents through both fistula and caecostomy openings. An emulsion, consisting of two grains of phenolphthalein with two drachms of castor oil in half an ounce of "Petrolagar", was given thrice daily in conjunction with the daily administration of a soap and water enema followed by irrigation of the colon with four or five pints of normal saline solution through the caecostomy opening.

Although slight infection of the abdominal incision occurred, this was firmly healed on the eighteenth day. The patient was discharged in excellent condition on April 30, 1932, with the fistula healed.

An abscess (? stitch) which had discharged about half a drachm of pus through the centre of the scar in May, 1932, had led to the formation of a suprapubic sinus about half an inch deep. When seen on July 15 the patient was still irrigating the colon thrice weekly and the suprapubic sinus appeared to be slowly healing. The de Pezzer catheter fell out on August 20 and a week later the caecostomy opening was soundly healed. On September 14 the sinus was reduced to an ulcer the size of a BB shot and an eighth of an inch deep. When last seen on November 9, 1932, all wounds were soundly healed.

Enough has been written to convince the reader of the close relationship between plumbing and surgery.

NASAL SINUSITIS IN RELATION TO PULMONARY TUBERCULOSIS.

By CLIVE M. EADIE, M.B., B.S. (Melbourne), F.R.A.C.S.,
Honorary Surgeon to the Ear, Nose and Throat Department, Melbourne Hospital.

IN a series of 104 *post mortem* examinations of patients who had died in the Melbourne Hospital from various diseases, six were found to have well-marked signs of pulmonary tuberculosis. All the nasal sinuses were thoroughly examined, a frontal mirror and reflected artificial light being used to inspect each sinus as it was opened up. Dr. Wright Smith, Pathologist to the Melbourne Hospital, performed the general examination, and my grateful thanks are due to him for the reports of the findings in each case. From the point of view of general interest, I have included the other lesions found *post mortem* besides the pulmonary condition.

I found that of these six cases, four showed no macroscopic evidence of any infection in their nasal sinuses at all. In two cases there were signs of infection in one or more of these sinuses, but the pathological state found did not warrant the conclusion that there was any association between the two conditions under discussion. The following is a description of these six cases.

CASE XXIX.—R.W., a female, aged twenty years, died on September 23, 1930.

The general *post mortem* findings were: chronic pulmonary tuberculosis, miliary tuberculosis of lungs, pleural adhesions, pyopneumothorax, thoracoplasty (first stage).

On examination of the nasal sinuses the right and left sphenoidal sinuses had normal mucosa, no pus, fluid or *débris*. The anterior and posterior ethmoidal cells, the maxillary antra, and the frontal sinuses were normal.

CASE XLVIII.—G.W., a male, aged sixty-eight years, died on December 1, 1930.

The general *post mortem* findings were: chronic pulmonary (fibroid) tuberculosis, pleural adhesions, pyonephrosis, peri-nephric abscess, carcinoma of the bladder, secondarily affected lymph glands, colostomy.

Examination of the right and left sphenoidal sinuses, the anterior and posterior ethmoids, the maxillary antra, and the

frontal sinuses showed that all had normal thin white membranes. There was no pus, mucopus or fluid in the cavities.

Case XXVI.—C.E., a female, aged fifty-two years, died on September 4, 1930.

The general *post mortem* findings were: pulmonary tuberculosis with adherent pericardium, tuberculous ulceration of the ileum, tuberculous peritonitis and cirrhosis of the liver.

Examination of the nasal sinuses showed that the sphenoidal sinuses were large and equal in size. No sign of any infection was found. No inflammatory signs were found in the left anterior ethmoidal cells; the right anterior ethmoidal cells were normal; the posterior right and left ethmoidal cells were normal. The right and left frontal sinuses were normal. In the right and left maxillary antra there was a thin white membrane. No inflammatory signs were present. No pus, fluid or *débris* was found in the cavities.

Case LIII.—A male, aged fifty-four years, died on December 21, 1930.

The general *post mortem* diagnosis was: pulmonary tuberculosis, bronchopneumonia, bronchiectasis, cerebral abscess, possible pneumococcal meningitis, perinephric abscess.

Examination of the right sphenoidal sinus revealed a large cell with no inflammatory signs present. On the left side there was a small cell, normal in appearance. The right and left posterior and anterior ethmoidal cells showed no inflammatory signs. No inflammatory signs were found in the right and left sphenoidal sinuses or in the right and left maxillary antra. Both middle ears showed no inflammatory signs.

The following are the two cases in which some signs of infection were found in the nasal sinuses.

Case LXIV.—D.J., a female, aged forty-three years, died on January 24, 1931.

The general *post mortem* diagnosis was: chronic pulmonary tuberculosis, tuberculous suprarenal glands, Addison's disease.

Examination of the nasal sinuses showed that the sphenoidal sinuses had thin normal membranes. No pus or mucopus was present. The membranes of the anterior and posterior ethmoidal cells showed no marked pathological change. The frontal sinuses were both small. The membrane was thin and normal in appearance; no pus or mucopus was present. In the right and left maxillary antra the membranes showed some catarrhal thickening. There was no evidence of any acute or subacute exacerbation; no pus or mucopus was present.

Case CI.—J.P., a male, aged thirty-eight years, died on April 20, 1931.

The general *post mortem* diagnosis was: chronic pulmonary tuberculosis, empyema, hydatid of the liver.

Examination of the nasal sinuses showed that in the right sphenoidal sinus the membrane was infiltrated and rather œdematous. Yellow, thin pus was present in the cavity. The left was the larger sinus. It had no sign of any inflammation. In the right posterior ethmoidal cell there was some infiltration of the membrane. In the left posterior ethmoidal cell no abnormality was detected. The anterior right and left ethmoidal cells were apparently normal. In the right frontal sinus the mucosa round the entrance of the naso-frontal duct showed marked congestion. The rest looked quiet. No pus, fluid or *débris* was present. On the left side no abnormality was detected. In the right maxillary antrum the membrane was rather infiltrated and œdematous. Some thin seropus was present in the cavity. On the left side no abnormality was detected. The *post mortem* examination thus revealed a terminal infection of the right anterior and posterior groups of nasal sinuses.

On investigating these cases we notice that in the first four (XXIX, XLVIII, XXVI, LIII) in the list there were no macroscopic signs of any infection in any of the nasal sinuses, the mucosa being thin without any evidence of hyperplastic changes, in fact quite normal in appearance. Yet in all of them there were marked tuberculous changes in the lungs, mostly of a chronic nature. The tuberculous infection had been going on for some years. Had there been any infection of the nasal sinuses, which was either causing direct tuberculous infection of the lungs, or acting indirectly predisposing the lung tissue to tuberculous infection, one surely would have found some changes in the sinus mucosa.

Now considering the two cases (CI and LXIV) in which there were some signs of infection present. Firstly, in Case CI there was evidently an acute inflammation of the right maxillary antrum and the right sphenoidal sinus, the right ethmoid cells and right frontal sinuses showing some lesser inflammatory signs. The sinuses on the left side appeared to be unaffected.

The appearance of the membrane of the affected sinuses was not that of an old standing chronic nasal sinusitis in the stage of an acute exacerbation. It was more that of an acute infection arising towards the termination of the patient's illness. One could hardly regard this condition in any way as a cause of the chronic tuberculous lesion found in the lungs.

In Case LXIV we found that there was some catarrhal thickening of the mucosa lining the maxillary antra. No thin serous fluid, pus or mucopus was present. The appearance of the membrane was not such as one finds in a chronic sinusitis, whether it be during the latent stage, or during an acute or subacute exacerbation. It was a mild catarrhal infection, such as is found in many patients, and so one is hardly justified in associating this mild catarrhal sinusitis with the chronic pulmonary tuberculous condition as cause and effect.

One realizes that the number of cases recorded here is very small, but it does seem to indicate that the association of infection and suppuration of the nasal sinuses with pulmonary tuberculosis is, to say the least, not a usual thing. In fact, considering these *post mortem* cases, and clinical observation of many hundreds of living patients, one is more inclined to believe that people with chronic nasal sinusitis do not often develop pulmonary tuberculosis, and conversely, those with chronic pulmonary tuberculosis do not often have any chronic infection of their nasal sinuses. The nasal sinuses rarely show any signs of tuberculous infection, either primary or secondary.

Reports of Cases.

THE "DOUBTFUL VERTEX": SIX CASES.[1]

By John S. Green, M.D. (Melbourne), D.G.O. (Dublin),
Honorary Obstetric Surgeon, Women's Hospital, Melbourne.

The cases here reported demonstrate various considerations in the management of the doubtful vertex case, including:

(i) Incompetence, tedious labour.
(ii) Disproportion.
(iii) Contracted pelvis.
(iv) Induction.
(v) Trial labour.

Five of the patients have been successfully delivered and the sixth patient is used to demonstrate the clinical investigation of a doubtful or border-line case.

[1] Read at a meeting of the Victorian Branch of the British Medical Association on July 20, 1933.

Case I.

Primipara, Incompetence, Tedious Labour.

A.W., aged twenty-seven years, was at term in her first pregnancy. She was in labour on admission to hospital and the head was fixed in the right occipito-anterior position. The membranes were ruptured and the os was dilated to one finger. Investigation showed the pelvic measurements as 25 : 27 : 21 centimetres and the fundus 27 centimetres.[1]

Progress was slow, but after forty-eight hours the os was nearly fully dilated, the head, though fixed, was not yet low in the pelvis, and the fœtal heart rate was 180 per minute. Fourteen hours later the baby, weighing 3·65 kilograms (eight pounds two ounces), was delivered easily by forceps.

In this case the pelvic measurements were not too small and the baby not too large, but the uterine mechanism was incompetent. One condition for forceps delivery, "os fully dilated", was present on the third day, but in the absence of maternal distress delivery was not attempted until the second condition, "head advanced", appeared on the fourth day. The fœtal heart varied considerably, but this did not precipitate interference and although the fœtal heart rate was at one stage 180, the baby was delivered in good condition over twenty-four hours later.

Case II.

Previous Still-Born Child, Disproportion, Induction.

M.A., aged twenty-six years, lost a 4·5 kilogram (ten pound) baby two years ago; the baby died just after a forceps delivery. In the second pregnancy investigation in the ante-natal clinic showed that the pelvis measured 24 : 26 : 19 centimetres. The fundus measured 27 centimetres when the pregnancy had advanced to thirty-seven weeks, and induction was decided on. The fœtus was in the right occipito-anterior position and the head was not fixed. The Johns Hopkins method was used with a rapid onset of labour and an easy delivery of a baby weighing 3·57 kilograms (seven pounds fifteen ounces).

In this case the pelvic measurements are slightly below normal, but the pelvis could not be classified as small. Disproportion due to a large child was the cause of still-birth. In the second pregnancy, induction when the fundus was 27 centimetres gave "an average child for an average pelvis", and an easy delivery resulted. It will be noted that the time for induction was decided by the size of the fœtus as represented by the height of the fundus and not specially by the period of the pregnancy.

Case III.

Previous Still-Born Child, Contracted Pelvis, Limited Trial.

V.S., aged twenty-five years, after thirty hours in labour, was delivered by forceps of a 2·47 kilogram (five and a half-pound) child, which died shortly after delivery.

In the second pregnancy investigation in the ante-natal clinic showed that the pelvis measured 22 : 26 : 17 centimetres and the case was noted as "probable Cæsarean after limited trial of labour". The patient was allowed to come naturally into labour at full time. After the labour had progressed for some time, the head was found to be well in the pelvis and an easy delivery of a baby weighing 2·56 kilograms (five pounds twelve ounces) resulted after about twelve hours' labour.

In this case the measurements are so diminished as to justify the diagnosis of a positively contracted pelvis. While induction is used, and should be used, to minimize disproportion, it is wiser with a frankly contracted pelvis to let the patient come into labour naturally and to allow a limited trial. The intention in this case was to do a Cæsarean section if developments were unsatisfactory after a limited trial, but the case fortunately demonstrated two points: (i) Nature's frequent provision of a small child for a small pelvis; (ii) the patient's greater efficiency as a *multipara*.

[1] To preserve sequence, the pelvic measurements are compacted. Thus 25 : 27 : 21 centimetres represents: Interspinous, 25 centimetres; intercristal, 27 centimetres; external conjugate, 21 centimetres. The height of the fundus is the direct caliper measurement from the *symphysis pubis*.

The considerations in management of the above cases will be accepted in the main, but the opinion as to induction in the *primipara* varies greatly. The following two cases are put forward as suggestive.

Case IV.

Disproportion, Cæsarean Section, Inductions, Cæsarean Section.

D.W., aged thirty-five years, was in her fifth pregnancy, and investigation showed the pelvic measurements as 20 : 25 : 18 centimetres. The first four confinements are briefly tabulated.

First: Albuminuria, still-born, macerated child.

Second: Cæsarean section after thirty-six hours' labour. Baby 4·5 kilograms (ten pounds).

Third: Induction with vaginal delivery of a 4·0 kilogram (nine pound) baby.

Fourth: Induction with vaginal delivery of a 3·6 kilogram (eight pound) baby.

In the ante-natal clinic the patient was given the option for her fifth confinement of "induction or Cæsarean section and sterilization", and in due course was delivered of a baby weighing 5·06 kilograms (eleven pounds four ounces) by Cæsarean section.

The conclusion to be drawn is obvious. If induction was successful after the Cæsarean delivery, it is logical to suppose that the induction could have been used primarily and the Cæsarean section avoided.

Case V.

Primipara, Disproportion, Induction.

M.M., aged twenty-six years, was thirty-nine weeks in her first pregnancy. Her pelvic measurements were 24 : 26 :20 centimetres and the fundus was 27 centimetres. The head was not fixed and was lying in the right occipito-posterior position. Induction by the Johns Hopkins method was commenced; progress was slow but sure, and about thirty-six hours later a baby weighing 3·37 kilograms (seven pounds nine ounces) was delivered by forceps.

In this case the pelvis was not contracted, but the head was not fixed about a week before term. The 27 centimetre fundus suggested a well developed child, and induction was done on the proposition of "an average child for an average pelvis".

Case VI.

Primipara, Disproportion, Induction and Trial Labour.

The following case serves to demonstrate simple methods of measurement and palpation, particularly in relation to the size of the pelvis, the size of the baby and the fit of the head in the brim of the pelvis.

E.B., aged nineteen years, was forty-one weeks in her first pregnancy. She had just come under observation in the ante-natal clinic and the pelvic measurements were 25: 27 : 18 centimetres and the height of the fundus 28 centimetres. The head was in the left occipito-transverse position and was not fixed.

This case is similar to the previous one, except that the optimum time for induction has possibly passed. The labour will be started by induction and the termination of the labour will depend on the progress made.[1]

Reviews.

BIOLOGICAL CHEMISTRY FOR THE CLINICIAN.

A VERY valuable contribution to the literature of the subject is to be found in the volume entitled "Biochemistry in Internal Medicine", by Trumper and Cantarow.[2] In

[1] Induction by the Johns Hopkins routine was commenced at 6 a.m. on July 22, 1932, and at 4 a.m. on July 23, 1932, a baby weighing 3·46 kilograms (seven pounds twelve ounces) was delivered naturally.

[2] "Biochemistry in Internal Medicine", by M. Trumper, Ph.D., and A. Cantarow, M.D., with foreword by E. H. Funk, M.D.; 1932. Philadelphia and London: W. B. Saunders Company; Melbourne, Brisbane and Christchurch: James Little and Son. Royal 8vo., pp. 454, with illustrations. Price: 36s. net.

recent years laboratory manuals of biochemistry have included much highly technical matter which it is difficult for the non-laboratory worker to appreciate. On the other hand, the advances in physical chemistry which are described in journals devoted to this subject are frequently not accessible to clinicians.

The authors of the book under review have shown considerable skill in selecting from a vast literature those details of biochemistry which have a clinical application. They state in their preface that "the essential function of the laboratory is to supply the clinician with information which will complement that which he may obtain by other methods. Each patient presents a problem which cannot possibly be appreciated on the basis of dissociated laboratory studies. However, in order to take full advantage of the findings of biochemists, the clinician must have a clear understanding of the significance and limitations of the results of laboratory investigation. This must be based upon an appreciation of the biochemical and physiological factors involved in the preservation and alteration of tissue and organ function, for it may be stated, more truly than ever before, that physiology is the handmaid of medicine." With these statements we heartily agree.

In attempting to supply the clinician with such information, Trumper and Cantarow have considered in turn the metabolism of carbohydrate, protein, cholesterol, chloride, calcium and phosphate. They then discuss the acid-base balance, respiratory exchange, *diabetes mellitus*, renal function, hepatic function, the chemical investigation of gastric function, pancreatic function, biochemical changes in pregnancy and lactation, cerebro-spinal fluid, transudates, exudates and œdema fluid. It is a pleasure to state that these subjects are treated in a concise, yet illuminating, manner. Let the chapter on calcium metabolism be taken as an illustration, the question of absorption and elimination of calcium is first considered, then the nature of blood calcium is discussed. This is followed by factors involved in the maintenance of the normal calcium level and partition involving an interesting discussion of the influence of the parathyreoid hormone, vitamin *D*, ultra-violet radiation and other factors. The causes of hypercalcæmia and hypocalcæmia are next reviewed, followed by short sections on alteration in distribution of calcium, abnormal urine calcium, and, finally, abnormal fæcal calcium. All these details are presented with excellent judgement.

In the chapter on gastric function it would be well to indicate in the accompanying graphs that the gastric acidity usually falls temporarily as a result of the diluting effect of the ingested meal. Although the authors have deliberately avoided giving a bibliography, this seems an unfortunate omission. Scattered throughout the book are references to the work of various authorities, and to add a short bibliography giving the exact source of such information would greatly add to what is otherwise an excellent publication and one deserving of widespread popularity.

BREAST FEEDING.

"THE BREAST-FED BABY IN GENERAL PRACTICE" is the title of a sensible little book by L. G. Housden.[1]

Dr. Housden has made a careful study of his small patients, their relatives and nurses, and his case records illustrate his points most ably. He writes with a sense of humour, which makes the study of the book a pleasure.

He points out that babies are primarily normal, healthy beings, but that their constitutions are often spoilt early in life by unnecessary drugs and artificial feeding. He quotes statistics to show that the breast-fed children, especially of poorer people, have a much greater chance of life and health than the bottle-fed ones.

He gives as the two important rules of breast feeding: (i) To preserve the breast milk, the breast must be emptied at regular intervals; (ii) to preserve the baby, the baby must be filled at regular intervals. He shows that if infants are test fed, they will often be found to be swallowing scarcely anything, though apparently sucking happily. In such cases breast feeding is often maintained by the milk being stripped off and fed to the child with a spoon.

He shows that an irregular gain in weight does not necessarily call for complementary feeding, and infrequent motions in otherwise healthy children do not necessitate purgatives.

Dr. Housden has carried out a series of investigations as to whether injections of breast milk act as a galactogogue, and on the whole his results are most promising.

The last chapter contains a full list of drugs that are secreted by the milk, and there is also a useful chapter on the management of premature infants.

This book should be read by all those really interested in the subject.

[1] "The Breast-Fed Baby in General Practice", by L. G. Housden, M.B., B.S.; 1932. London: H. K. Lewis and Company, Limited. Crown 8vo., pp. 127.

THE LOCALIZATION OF DISEASE.

THE reasons why pathological changes should be confined to this or that situation in the courses of various diseases are necessarily of great interest and importance to all who practise our profession. What is the significance of the distribution of a skin rash? Why does the *Staphylococcus aureus* or the streptococcus establish itself in the periosteum or the medulla of bones? Why is pigment deposited in the skin in some places more than in others? Why does a local infection not necessarily spread, and why, when bacteria enter the general circulation, may we find that they are arrested in certain localities and do not necessarily seed all structures alike? These are questions that we frequently ask ourselves, and Dr. Harold Burrows, in "Some Factors in the Localisation of Disease in the Body", helps us to understand better the various agencies at work and thus enables us, if not to supply full answers to the questions, at least to grasp some of the principles involved.[1]

The work opens with a consideration of the permeability of the capillary endothelium, and more particularly of the reticulo-endothelium, and the author points out how, during inflammation, this permeability is greatly increased so that negatively charged particles, such as colloids, certain dyes, particulate matter, bacteria, viruses and so on, can readily pass through the endothelial cells and so become aggregated in the inflammatory site. Thus consecutive chapters deal with the permeation of the capillary endothelium by normal proteins, by foreign proteins, by dyes and fine inorganic particles, by natural pigments, by the treponema of syphilis, by bacteria and by viruses, with a final chapter in Part I on the application of the principles considered to the problems of cancer. Eight coloured plates of the reactions of animals under various conditions to the injection of Indian ink or vital dyes well illustrate the matters discussed. In Plate I, Indian ink particles introduced intravenously are shown located in the readily permeable reticulo-endothelium of the liver, spleen and bone marrow, and also in the neighbourhood of an inflammatory reaction, thus showing the increase of the permeability of the endothelium of the capillaries as a result of this reaction.

In Part II the author discusses the factors that may increase the permeability of the capillary endothelium, and also considers the retention of colloids and other substances, including viruses and bacteria in an inflamed tissue.

In Part III are discussed the inflammatory barrier, persistent endothelial permeability and the therapeutic considerations that naturally arise.

We have read this work with pleasure and profit and believe that many in general or special practice will also find it of interest and value. The price, it will be noted, is moderate.

[1] "Some Factors in the Localisation of Disease in the Body", by Harold Burrows, C.B.E., F.R.C.S.; 1932. London: Baillière, Tindall and Cox. Medium 8vo., pp. 311, with eight illustrations. Price: 15s. net.

The Medical Journal of Australia

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All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for publication, are invited to seek the advice of the Editor.

MINERS' NYSTAGMUS.

ALTHOUGH the condition known as miners' nystagmus was first recognized by Gillet, of Sheffield, in 1854, it has become the subject of much investigation and controversy only since the enactment of measures to compensate men for disability arising out of or during their employment. In 1922 and again in 1923 the Medical Research Council of the Privy Council of Great Britain issued special reports dealing with the subject; these were discussed in this journal in the issues of September 9, 1922, and October 10, 1925. The definition of the condition given in the first report was: "The disease known as miners' nystagmus, whether occurring in miners or others and whether the symptom of oscillation of the eyeballs be present of not." The main finding of the committee issuing the first report was that the chief symptom of the disease, involuntary oscillation of the eyeballs, was caused by an insufficiency of the light reaching the eye of the miner while at work, and that of all the measures for prevention the most important was to secure for the miner adequate illumination.

Since the first report was issued there has been no reduction in the incidence of miners' nystagmus and the Medical Research Council has, therefore, issued a third report.[1] The committee in this instance is representative of the earlier body, but includes several members who were selected for their ability to give advice on the psychological and statistical aspects of the problems. The personnel of the committee is as follows: Professor M. Greenwood, F.R.S. (Chairman), Professor E. L. Collis, Dr. Millais Culpin, Dr. J. S. Haldane, F.R.S., Dr. L. Isserlis, Dr. T. L. Llewellyn, Sir J. H. Parsons, F.R.S., Sir David Munro (Secretary). The present report must be regarded as a continuation of the two previous reports. Those who would study the subject in its entirety are advised to read all three documents. The present report is short and will give those who have to deal with affected miners an insight into the present state of knowledge and will show them that correct diagnosis and adequate treatment cannot be attained without attention to detail and careful weighing of evidence.

The committee refers to some of the hypotheses which have been advanced in explanation of miners' nystagmus: that it may be the result of errors of refraction, of abnormal posture causing fatigue of the eyeballs, of spectral quality of light supplied, of glare and of poisons such as mine gases and alcohol. They hold that these do not rest on sufficient evidence to justify alteration of the original committee's conclusions, that the fundamental cause of miners' nystagmus in the narrower sense of the term (that is, oscillations of the eyes) is the low illumination under which miners in general work. Some interesting figures are given in regard to illumination. Under dim illumination the peripheral parts of the retinal field are used. The use of peripheral vision is characteristic of dark adaptation; when dark adaptation is enfeebled, the individual suffers from night blindness, and night blindness is one of the commonest early symptoms of miners' nystagmus. It has been found that vision changes over from central to peripheral when the intensity of light reaching the eye is 0·0073 of a milliambert or 0·0068 foot-candle; this finding is presumed to hold good under conditions of the coal mine. The amount of light reflected into the

[1] "Third Report of the Miners' Nystagmus Committee", The Medical Research Council of the Privy Council. Special Report Series, Number 176. 1932. London: His Majesty's Stationery Office. Pp. 36. Price: 9d. net.

eye determines the clearness of vision and coal absorbs a large percentage of incident light. The committee holds that if illumination over the immediate working area were raised to 0·1 foot-candle maintained throughout the shift, the incidence of miners' nystagmus would be greatly diminished. The ultimate goal to be aimed at is a constant illumination of 0·25 foot-candle on the coal face.[1] The fact that diminution in the incidence of nystagmus has not always followed attempts to improve illumination, is largely due, in the opinion of the committee, to the use of ineffectual lamps which neither give a sufficient light at the commencement of a shift nor maintain their defective light throughout it.

The main interest in the report lies in the relationship of psychological factors to disability. The psychological factors are mainly responsible for the need for compensation. There is a latent or early stage in which oscillation of the eyes is not noted. Later on, when oscillation has occurred, as the psychological symptoms make their appearance and increase in severity, the oscillation of the eyes becomes less and may disappear. This is shown by analysis of several cases made by Professor Culpin and published as an appendix to the report. It is pointed out that in civil practice and under war conditions it has been commonly recognized that a bodily manifestation of hysteria can protect a patient against other symptoms, and that when the hysterical symptom, such as a tremor, has disappeared, severe anxiety or obsessional symptoms may occur spontaneously or as a result of treatment. Cases of miners' nystagmus are, therefore, put into the following general scheme:

A. Oscillation with no disability.

B. Oscillation with subjective movement of objects. This appears to be a recoverable stage and may be organically determined. On the other hand, the added symptom may behave as a hysteria and for a time protect against further developments.

C. The unfolding of further psychologically determined symptoms—hysterical, anxiety or obsessional—with the oscillation now behaving as an hysterical tremor and tending to disappear.

D. The disappearance of oscillation and the complete development of a psychoneurosis.

[1] A foot-candle denotes the illumination derived from a source of one standard candle at a distance of one foot. The intensity of illumination varies inversely with the square of the distance from the source of light: thus one candle at one foot, four candles at two feet, and nine candles at three feet all give equal illumination.

Another interesting aspect which is mentioned and which might be discussed at length is the consideration of those men among the general population who are the subjects of psychoneurotic symptoms or tendencies. These men might break down in the mines without the question of nystagmus arising. Some might find in the eye troubles an escape from their other symptoms, for the substitution of mental symptoms for organic is reversible. Professor Culpin states that whether there is a disability independent of any psychological element may be left an open question. He also adds that there is sufficient evidence to show that the psychological effect of "darkness made visible" is a likely factor in the stimulation of a pathological fear of the dark.

From the practical point of view the treatment of the condition calls for more discussion than can be allowed in this place. The committee thinks that the psychological factors are those which need most attention. The condition is not so hopeless as some Australian practitioners have believed it to be. The committee holds that the first essential to a satisfactory handling of the "disease *certified* as Miners' Nystagmus" is a better understanding of its nature and especially of its analogy to what was known as shell shock. The first step is to combat the belief that the psychoneurotic symptoms can be a direct result of oscillation of the eyeballs. The final recommendation is as follows:

> The Committee are strongly of opinion that the practical treatment of the disease from an administrative point of view should consist in the elimination of a hopeless dependence on compensation by the provision of opportunities for work of some kind, the end in view being complete restoration to full work underground, under conditions of proper illumination, even if this has to be preceded by a period of work in daylight.

Miners' nystagmus in Australia is not different from miners' nystagmus in other parts of the world. Treatment is not merely a matter of ordering a man to stop his work at the "face" and seeing that he obtains compensation if he is entitled to it. The Medical Research Council's committee believes that the true significance of the various symptoms presented by sufferers from this complaint can be elucidated only by medical men with requisite training. Australian practitioners, particularly

those in coal mining areas, must study the question. If they have not begun to study it, they will find this report extremely useful as a beginning; if they have devoted time to it, they will find the report helpful, particularly that part devoted to an analysis of cases. Their greatest difficulty will always lie with the miner, rare perhaps, who wants something for nothing and who is prepared to make the most of his disability.

Current Comment.

FOCAL INFECTIVE PROSTATITIS.

SEPTIC tonsils, apical (dental) and other septic foci are recognized as the cause of such remote symptoms as those due to arthritis, fibrositis and neuritis. Treatment of these symptoms is futile while the septic focus persists, but at the same time the medical mind must not be obsessed and the matter must be regarded in proper perspective. The fact that many teeth and tonsils may have been removed unjustifiably and without benefit to the patients does not vitiate the truth of the fundamental principle. To teeth and tonsils must be added, in the male, the prostate gland as a site of focal sepsis. A prostatic focus may be the cause of arthritis and other remote manifestations. On the other hand the prostate may be infected from focal sepsis in other parts, such as teeth or tonsils. Chronic prostatitis and prostatic abscess were long considered to be mainly the result of gonorrhœa. This view is not at present generally held. Authorities, however, differ widely as to the percentage of cases due to Neisserian infection. Gonococci appear in the prostatic fluid in diminishing numbers for the first three years following prostatic involvement due to gonorrhœa. After that time gonococci are rarely found unless the patient has been reinfected. As a rule gonococci die soon, but they induce a lowered resistance favouring infection by other organisms. Organisms isolated from infected prostates include *Streptococcus viridans* (and others), *Staphylococcus albus* and *aureus, Bacillus coli, Micrococcus catarrhalis* and diphtheroid bacilli. In the non-venereal form infection may be long dormant and may be discovered only by slight pyuria. Prostatic abscess or chronic prostatitis has followed abscess of the nose, osteomyelitis of the finger, typhoid fever, paratyphoid fever, influenza, pneumonia, measles, septicæmia and other infections. But prostatic infection is not necessarily due to the organism causing the systemic infection from which the patient is suffering. Remote symptoms of prostatic infection include polyarthritis in various regions, sciatica and widespread neuromuscular pains. Ocular symptoms may also be prominent, particularly in non-gonorrhœal cases, in which the cornea and uvea are most often implicated; in gonococcal cases the cornea and iris are most often affected. When a metastatic inflammation subsides after elimination of a possible source of infection, it is likely that infection at the source was the actual cause of the inflammation, but inflammation may persist after removal of the focus of infection, because tissue resistance has been lowered and inflammation may be perpetuated by toxins or organisms from elsewhere.

P. S. Pelouze[1] asserts that at least 72% of all males with focal infective symptoms have prostatic infection and cannot be made permanently symptom-free while it persists. At least 35% of all males over the age of thirty-five have such a focus of infection. The infected prostate has almost no relation to gonorrhœa. Pelouze reports one hundred patients with prostatic infection. Of these only two had had gonorrhœa within twenty years, and only seven had ever contracted gonorrhœa. Prostatic infections are rarely primary. Pelouze points out that there is a definite association between dental or tonsillar infection and involvement of the prostate. While these foci in the teeth or tonsils persist it is rare for prostatic infection to be permanently cured. He goes on to state that prostatic infection is seldom or never associated with infections of the facial sinuses; it is occasionally caused by influenza or other acute infection and perhaps gastro-intestinal or gall-bladder infections may play a part. The opinion of R. Pemberton is quoted, that there must be an initial systemic factor which probably antedates the determination of all these infective foci and sensitizes the individual to their toxins. In Pelouze's opinion, not only existing dental infections must be dealt with, but watch must be maintained for other tooth-root infections. Dental radiograms should be taken at intervals for two years. At least 64% of those having teeth extracted for infection show later infections of other teeth. Tonsils also should be examined; apparently innocent tonsils may be infected. If the tonsils and some teeth are removed and the prostatic infection clears up and then recurs, other dental infections must be sought. If not removed, the tonsils are suspect when no further dental infection can be ascertained; or the gall-bladder may need investigation. If all these investigations yield no definite information and if there is no change in the remote symptoms following prostatic treatment, the gastro-intestinal tract should be examined, as the prostate is probably not concerned.

Pelouze goes on to discuss prostatic massage. He points out that prostatic massage may be followed by pyrexia and increase in joint pains. This is to be expected from clinical experience with other septic foci. The series quoted by Pelouze is too small to warrant any generalization as to the incidence of prostatic infection, but his paper is useful because involvement of the prostate is often overlooked by medical practitioners who do not make a special study of genito-urinary disorders.

[1] *The American Journal of the Medical Sciences*, August, 1932.

Abstracts from Current Medical Literature.

THERAPEUTICS.

Detoxication with Special Reference to Sodium Ricinoleate.

T. H. Rider (*The Journal of Laboratory and Clinical Medicine*, October, 1932) has investigated the comparative detoxifying activities of sodium ricinoleate and the other products found in a crude castor oil soap. He points out that comparatively little work has been done upon the detoxifying power of pure soaps, so that no quantitative relationships have been established. In most cases the soap is referred to merely as a soap of the oil from which it has been prepared, and even where reference is made to pure soaps, the purity is questionable. The author doubts whether a truly pure sodium ricinoleate has ever before been prepared. Detoxication is a more or less general property of soaps, though their detoxifying powers vary greatly. Sodium ricinoleate is the most potent soap as a detoxifying agent, but he has not found any of the methods for the preparation of a pure sodium ricinoleate to be satisfactory. The difficulties are discussed and the author indicates his own method of preparing a chemically pure sodium ricinoleate, the details of which he has already published. An attempt has been made to determine the effect of concomitantly occurring impurities upon the detoxifying activity of sodium ricinoleate of various degrees of impurity. Comparative experiments were made, tetanus toxin mixed with crude soap, with partially purified soap and with chemically pure sodium ricinoleate being used. The percentage composition of the crude soap was approximately: sodium ricinoleate, 90; sodium oleate, 1; sodium linoleate, 6; sodium stearate, 1; sodium dihydroxystearate, 2; and of the partially purified soap: sodium ricinoleate, 93; sodium oleate, 1; sodium linoleate, 6. Chemically pure samples of the sodium salts of the contaminating fatty acids were prepared with a view to studying their detoxifying power. Only pure sodium oleate and sodium linoleate (in solutions of a concentration of 1% and 0·5%) could be proved to have any detoxicating power. It is possible that the concentration of the soap, not the absolute amount present, is the determining factor in detoxification. One-tenth of a milligramme of the tetanus toxin available was taken as the minimum lethal dose for a guinea-pig of 250 grammes weight. One hundred times this amount was dissolved in one cubic centimetre of the fluid to be injected. Concentrations of soap solutions of 0·1%, 0·2%, and 0·25% were sufficient to show the superiority of sodium ricinoleate, but only afforded partial protection. With 0·3% concentration, when the chemically pure sodium ricinoleate was used, all the guinea-pigs were alive and showing no signs of tetanus at the end of eight days, but when the crude soap was used, all showed signs of tetanus and two-thirds of them died between the seventh and eighth days. Sodium oleate and sodium linoleate in 1% solution both gave perfect protection. The increase in activity secured by purification may be only slight, but there are manifold other advantages obtained.

Peptic Ulcer.

Z. Martin (*Annals of Internal Medicine*, November, 1932) describes the results of treatment of 95 patients with peptic ulcer by injections of milk protein. The patients generally remained at work on their usual diet. Diagnosis was assisted by radiological examination and gastric analyses. Six intramuscular injections of a non-specific milk protein, "Aolan", were given on alternate days. If symptoms decreased slowly, more than six injections were given; ten cubic centimetres were injected each time. Relief of symptoms was noted frequently after two or three injections. Little change was noted in the X ray films as a rule, though spasm was sometimes decreased. X ray evidence of ulcer was still present in some patients clinically well. Immediate results of treatment were usually better than late results.

Acetyl-Salicylic Acid.

R. W. Lamson and R. Thomas (*The Journal of the American Medical Association*, July 9, 1932) discuss allergic response to acetyl-salicylic acid. Four cases are described in which an asthma cure, "M. Matte's German Asthma Powder", containing caffeine and acetyl-salicylic acid, was ingested. In each instance there was a history of asthma, and also of sensitiveness to acetyl-salicylic acid. The ingestion of the preparation in these four cases was followed in two by severe dyspnœa, in one by dyspnœa and unconsciousness, and in the last case by dyspnœa and death within half an hour. It appears that these effects were due to an abnormal effect of acetyl-salicylic acid, to which the persons affected were known to be sensitive.

Small Calomel Doses in Treatment of Neurosyphilis.

Buschke and Boss (*Deutsche Medizinische Wochenschrift*, November 11, 1932) state that calomel is very useful in the treatment of neurosyphilis (excluding tabes and *dementia paralytica*), for its effectiveness far surpasses that of "Salvarsan" and bismuth preparations. Insoluble mercury salts are better than the soluble salts, since their effect is more lasting. If small doses of calomel (five to ten injections of 0·025 gramme at intervals of three to five days) are given, severe complications of calomel poisoning (colitis, stomatitis *et cetera*) do not occur. All patients should be thoroughly examined before treatment. Early acute syphilitic meningitis and acute meningo-neuritis respond well to calomel injections, especially if, as often occurs, "Salvarsan" has been given previously. In observations on fifty cases over a three-year period, calomel injections, followed later on by "Salvarsan" or bismuth or mercury inunctions, have given good results, especially in the acute meningitic cases; also in chronic meningitis. In brain tissue lesions calomel has rendered the process stationary.

Damage to the Bladder Mucosa in Bismuth Therapy of Syphilis.

Grundmann (*Münchener Medizinische Wochenschrift*, August 19, 1932) states that in bismuth therapy albuminuria and cylinduria are well known complications. Very rarely inflammation of the urinary tract, especially of the bladder, occurs and may account for albuminuria. He quotes Engelhardt, who found on cystoscopy in nine cases swelling and reddening of the bladder mucosa; he states that cases have occurred in which pin-point deposits of bismuth have been seen in the mucosa. Subjective symptoms of hæmorrhagic cystitis may occasionally occur before signs of kidney damage or stomatitis appear.

Salt-Poor Diets.

Baumgartener (*Münchener Medizinische Wochenschrift*, October 7, 1932) states that he has made the following observations during the past four years in using salt-free diets. (i) No benefit occurs in those unspecific inflammatory diseases with a marked leucocytosis. (ii) Marked benefit often occurs in those disease in which an exudation is present associated with a poor or no leucocytic response. As recognized clinically, the diminution of the exudate often runs parallel with the lowering of the salt intake. In every tuberculous focus at some stage of its development a more or less perifocal exudate occurs and the author therefore recommends a salt-poor diet in these cases. Good results have also been seen in lupus, tuberculous effusions in joints and tuberculous cystitis.

Fever Therapy in Chronic Non-Specific Arthritis and in Bechterew's Disease.

Horn (*Münchener Medizinische Wochenschrift*, November 25, 1932) writes that early and intensive treatment in chronic non-specific arthritis and Bechterew's disease is important, since very frequently these conditions give rise to joint deformities. The many different treatments ordered indicate that even today clinicians are unable to treat them successfully or to cure the lesion. Treatment is mainly symptomatic. Protein shock therapy in many cases does not fulfil the expectations demanded of it, but it is often very useful. In his series of cases he used "Pyrifer" (prepared from pyrexia-causing bacteria, non-

pathogenic). Injection with this product causes a rise in temperature similar to that of malaria. Fever therapy acts in the following way: (i) It converts a chronic inflammatory process of the joints into an acute process, and thus there is a better tendency to healing as a result of the acidosis. (ii) It supports and increases the natural powers of resistance by a marked marrow irritation. (iii) It causes a marked outpouring of leucocytes. He quotes two patients with Bechterew's disease suffering since 1924, one of whom had almost been bedridden since 1927; marked improvement and freedom from pain occurred after nine injections of "Pyrifer". In another case of primary *polyarthritis rheumatica* of elbow and shoulder joints, five injections of "Pyrifer" produced a definite improvement.

NEUROLOGY AND PSYCHIATRY.

Lumbar Puncture and Sedatives.

SAMUEL H. EPSTEIN AND GEORGE M. LOTT (*The Journal of Nervous and Mental Disease*, December, 1932) conclude that "Sodium amytal" is the best sedative to administer to patients prior to lumbar puncture. Practically all the sedatives and hypnotics have been used to prevent or mitigate the after-effects of lumbar puncture. In psychotic conditions particularly, where lumbar puncture is a well-established diagnostic procedure, it is necessary to use a suitable sedative in order to allay that restlessness and fear which make it so difficult for the patient to cooperate with the physician. These authors conclude from their research that such sedatives as "Sodium amytal" and "Nembutal" most generally facilitate the operation of lumbar puncture, especially in neurotic and psychotic patients. They recommend 0·36 gramme (six grains) of "Sodium amytal" or 0·18 gramme (three grains) of "Nembutal". Within an hour of such administration the patients are generally in a calm, somnolent state, which may last for a period of eight hours and from which the patient wakes without any unpleasant memories. In addition, the post-lumbar puncture reactions are diminished, although not altogether eliminated.

A Salt Substitute in an Epileptic's Diet.

HOLTHAUS (*Münchener Medizinische Wochenschrift*, September 9, 1932) states that in treating epileptics with bromides and "Luminal", a salt-free diet is often of very great value, for the number of fits is decreased. On the addition of salt to the diet, fits often recur, and this has also been proved on giving sodium chloride intravenously. Many of the patients complained that a salt-free diet was unpalatable, caused loss of appetite and in a few even nausea and vomiting. A preparation "Curtasal" (composed of magnesium, sodium and potassium salts, having a slightly alkaline reaction and an intense salty taste) is an excellent substitute for salt and can be added to all diets without increasing the number of fits. Volhard has also used up to three grammes of this salt per day in the treatment of patients with cardiac and renal conditions without causing any damage. "Curtasal" is therefore very valuable where a salt-free or salt-poor diet is desired without altering the palatability of the diet.

Ear, Nose and Throat Sepsis in Mental Disease.

T. A. CLARKE (*Journal of Mental Science*, July, 1932) believes that septic conditions of the ear, nose and throat play a direct part in the production of mental disorder. The author considers in detail the various ways in which the influence on the mental functions may act, as by: (i) passage of organisms or toxins to the cerebrum by virtue of proximity; (ii) circulating toxins, specific or non-specific, acting directly on the cerebrum or affecting the endocrine system; (iii) sensory disturbance; (iv) obstruction of respiration and impairment of oxygenation of the blood. The highest incidence of tonsillar sepsis occurred, in this author's series of cases, among those suffering from *dementia præcox*. He contends that the incidence of ear, nose or throat infection in mental disease is considerable and may predispose to or exaggerate the development of psychotic states.

Brain Tumours and Cellular Changes in the Cerebro-Spinal Fluid.

H. HOUSTON MERRITT AND MERRILL MOORE (*Journal of Neurology and Psychopathology*, October, 1932) comment on the cellular changes in the cerebro-spinal fluid associated with cerebral tumour; and they draw attention to the presence of polymorphonuclear cells occurring in certain neoplastic conditions. They report fully upon two cases of glioma of the anterior portion of the *corpus callosum* and frontal lobes, which yielded a fluid turbid with polymorphonuclear cells upon lumbar puncture. They show that, although the findings of increased intracranial pressure with pleocytosis and normal sugar content usually indicate a septic focus within the brain or meninges, such findings are not inconsistent with certain types of brain tumour. They refer to a *glioblastoma multiforme* of the temporal lobe which had compressed the middle meningeal artery, producing an area of softening in the temporal lobe near the ventricle. They claim that the presence of polymorphonuclear cells is explained as being due to "drainage of cells from the area of symptomatic inflammation occurring around foci of necrosis close to the ventricles". The fact that this type of cell increase is not more generally found is due, these authors maintain, to the fact that lumbar puncture is seldom performed after a definite diagnosis of cerebral tumour is made, especially if associated with marked papilœdema; and the polymorphonuclear cells may be expected in such cases only when a recent hæmorrhage has occurred or an area of softening has developed.

Pernicious Anæmia with Mental Changes.

A. PINEY (*Journal of Neurology and Psychopathology*, October, 1932) claims that certain abnormalities of conduct may occur as prodromal symptoms in patients who develop pernicious anæmia. He further states that psychotic symptoms sometimes occur in patients whose blood picture shows but slight variation from the normal, and that improvement in the mental symptoms takes place when the hæmatological malady is cured. He recommends liver therapy. Piney contends that it might be profitable to examine the blood in all cases of mental disorder in which there is the slightest evidence of anæmia. There is no known mental state characteristic of pernicious anæmia; but it is doubtless true that this blood disorder may cause a profound disorder of the nervous system which is usually of a depressive or paranoid character.

Masturbation in the Ætiology of Psychotic States.

WILLIAM MALAMUD AND G. PALMER (*The Journal of Nervous and Mental Disease*, September and October, 1932) review the whole question of auto-erotism in relation to psychiatry. Their observations and conclusions were based upon the examination of five hundred consecutive patients admitted to the Iowa State Psychopathic Hospital. Fifty cases are presented in detail, and information is given under the headings of: (i) sex and civil conditions, (ii) age, (iii) evidence of constitutional peculiarities, (iv) type of disturbance, and (v) the relationship of the practice of masturbation to the type of mental disturbance. The conclusions reached are that in a certain number of mentally diseased persons the development of the disease is closely related to the practice of masturbation; that, quite apart from the effects of masturbation as such upon the personality development of the individual, masturbation has brought about conflicts clearly associated with the particular mental disorder; that the development of abnormal mental states showed no clear relationship to the continuation of masturbation in adult life; that mental deviations related to masturbation were commoner in men than in women, occurring in the former during adolescence and in the latter at the preclimacteric period. Masturbation as an ætiological factor in the production of abnormal mental states was found to predominate where there existed constitutional peculiarities as evidenced by poor heredity, where there was lack of proper sex education and misinformation regarding the alleged effects of masturbation. The authors claim that psychotherapy was found effective in the readjustment of a number of their patients.

Special Articles on Treatment.

(Contributed by request.)

VI.

THE GENERAL MANAGEMENT OF A PATIENT WITH EARLY PULMONARY TUBERCULOSIS.

At the outset it is necessary to have a clear understanding of the meaning of the phrase, "early pulmonary tuberculosis". It has, of course no relation to time or apparent duration of the disease. A patient's disease may be hopelessly advanced within a few weeks of the apparent onset of symptoms; on the other hand, he may still be an "early case" many years after the diagnosis has been established. It is, in fact, impossible to draw an arbitrary line separating the early from the more advanced cases of pulmonary tuberculosis. Some authors maintain, for instance, that once physical signs, especially râles, are detected, the case is no longer early.

Chest signs should certainly be such as to suggest that the lesion is localized to one, or at the most two, lobes, and cavitation should not be apparent. Some latitude may be allowed the patient, even if radiological evidence denotes more extensive infection. The finding of disseminate infiltration of both lungs radiologically demands, as a rule, a diagnosis of advanced disease. Evidence of toxæmia is, however, in most cases, the deciding factor; pyrexia, tachycardia and other evidence of severe toxæmia persisting despite complete rest, preclude a diagnosis of "early" disease, however scanty physical signs may be. Complications, especially tuberculous laryngitis, place a patient in the advanced class.

If a definition of "early" is demanded, the following may be offered. The patient's constitutional symptoms are slight, with evening temperature not above 37·2° C. (99° F.) and resting pulse rate below 90 per minute. Physical signs should be limited to one lobe or to the apices (for example, above the clavicles or spines of scapulæ) of both lungs. Criticism can be levelled against this definition, not least because it ignores radiographic evidence. But whatever is the accepted criterion of an "early case", be it remembered that the once famous Turban-Gerhardt classification is now obsolete, inasmuch as symptoms are more important than signs.

In view of what has been said above, treatment must be considered from two viewpoints. First, we have to treat a chronic inflammatory lesion of the lung; secondly, we must combat the toxæmia which is producing the patient's general symptoms.

In preparing a plan of treatment, the ultimate aim must be kept clearly in view, that is, to enable the patient to return to the occupation (though not necessarily, of course, to the mode of life) previously held by him. This may sound idealistic, and it will require modification in the individual case. But the fatalism of most laymen and of many doctors in regard to the outcome of tuberculosis is to be deplored. In the words of the great Robert Koch: "This is a conflict which we may enter with a surely founded prospect of success." Arrest of the disease process can be attained in the majority of early cases, given adequate treatment, though many factors operate to make this happy issue still relatively infrequent. Ofttimes it cannot be denied that the physician must accept a share of blame for the failure of the patient to withstand the ravages of the tubercle bacillus.

Just what percentage of pulmonary tuberculosis is actually diagnosed in the early stage it is difficult to state; probably considerably less than 50%. It is, of course, well known that some "early cases" go steadily downhill and die despite the most careful attention, while others with advanced disease live as invalids for many years.

Our first principle in treatment is rest. This is axiomatic, and yet it is more often honoured in the breach than in the observance. Rest is the basis of all treatment in tuberculosis. It is the correct treatment for the local lesion in the lung, and for the general toxæmia, and it may be the only treatment necessary. In the freshly diagnosed case many authorities demand an initial period of rest in bed of several months, if there is any evidence of toxæmia, as there usually is. This may be over-cautious, but there is no doubt that the importance of complete rest, both mental and physical, is not sufficiently appreciated in the early stages. No detail can be given here regarding rest and exercise in pulmonary tuberculosis, but the reader is urged to study carefully for himself, in one of the many excellent text books available, this aspect of treatment.

It is generally agreed that this "rest" period, whether complete or modified, is best carried out in an institution, usually a sanatorium. The patient often objects, but it is worth while explaining the benefits of a sanatorium to him. Disciplined treatment, with a regulated and vegetative existence, education for future life, and last, but not least, the community spirit found in a well-conducted sanatorium, these assets more than compensate for the temporary breaking of home ties. A minimum period of six months should be insisted on. More cannot be said on this, except to emphasize that the febrile patient should be treated in hospital and not sanatorium.

Should the patient for any reason be unable or refuse to go to a sanatorium, domiciliary treatment is necessary, either at home or in the country, in a chosen climate. In either alternative the patient should follow a strict routine prepared for him on sanatorium lines, under medical supervision. Here it is opportune to give two warnings. First, there rests a grave responsibility on the doctor who gives such advice as "you must go to the country for six months" or "you should get away to a dry climate" and leaves the rest to Providence. Should this patient be fortunate enough to find his disease quiescent at the end of this period (and this rarely happens), he has learnt no lessons for the future in case of a breakdown. More often he either deteriorates slowly or breaks down badly and enters the "advanced" class. In treating a patient with pulmonary tuberculosis, too much emphasis cannot be laid on the need for attention to details, which must, moreover, always be fitted to the individual case. There should be the same cooperation between medical man and patient as in diabetes. No encouragement is to be given to the request of the relatives that the patient be not told what is the matter. "No fool was ever cured of tuberculosis of the lungs" (Kingston Fowler), and the patient who cannot be told his diagnosis will never do any good.

Secondly, avoid the exaggerated value usually allotted to climate in the treatment of pulmonary tuberculosis. Other things being equal, a mountain climate is an important auxiliary, but it is for most patients safer to live in a comfortable suburban home than to rough it in a wind-swept mountain shack several miles from the amenities of civilization. Here again all factors must be carefully weighed before arriving at a decision in the individual case.

This brings us to the second line of attack, which is to insure an abundant supply of fresh air for the patient. As far as possible he should live in the open air, and sleeping out should be advised, providing a veranda can be found facing north or east. It need hardly be said that sleeping in a tent has little to commend it.

Little need be said concerning diet. Three good meals a day, with a well-balanced dietary, and perhaps additional milk till lost weight is regained, are all that is necessary. Give extra fat, if it is well tolerated, remembering that "fat-shy" patients as a rule do badly, but the days of "over-feeding" have passed. Nor have the salt-free and other special diets of German authorities proved of value in the pulmonary form of tuberculosis.

There is as yet no direct specific or therapeutic cure for tuberculosis. By the methods outlined above the individual slowly builds up his resistance by his own defences. Of various drugs and the like nothing will be said here, except that they have two main virtues: (i) By suggestion they encourage a hopeful attitude in the patient towards ultimate cure. (ii) They enable the physician to keep in touch with his patient.

Arising out of the first great principle of rest, consideration must be given to the recent development of local rest to the damaged lung, by what is known as "collapse therapy". Of the several varieties thereof, only the most important will be mentioned here, namely, artificial pneumothorax. This is now generally recognized as the greatest adjunct to the treatment of pulmonary tuberculosis which we possess. It is universally employed today and there is a growing tendency throughout the world to use it in earlier cases. Many authorities urge its use, in the unilateral case, as soon as a diagnosis has been made, no matter how limited the lesion. The "wait and see" policy is open to the objections that pleural adhesions may form or the lesion may become bilateral, both of which untoward events will prevent pneumothorax therapy. It is the experience of most who have used this treatment that whereas they have very rarely, if ever, regretted the inducing in a patient of artificial pneumothorax, they have often lamented that they "waited to see" and thereby lost the opportunity of carrying out this valuable treatment. There is scope for much argument here, but the following dictum will find general agreement as a middle course. If after, say, three months of careful routine treatment, the patient with a unilateral lesion is not making steady progress, artificial pneumothorax is indicated.

For further information regarding indications and technique of artificial pneumothorax, the reader is referred to text books on pulmonary disease, of which several are given below. Be it remembered, of course, that collapse therapy is only an accessory to, and not a substitute for, the general treatment already outlined.

There is not space to consider treatment with the modern gold salts, such as "Sanocrysin", "Solganal" and "Crisalbine". They have definite uses, and brilliant success can be won with them, but they are not often indicated in early cases.

Heliotherapy, to which our country is so admirably adapted, might be discussed at length. 'Twere safer to leave it here with the advice of Mr. Punch: Don't!

Symptomatic treatment cannot be touched on. Most text books give this in full detail.

In conclusion, mention should be made of the importance of the psychical side of our treatment. An optimistic outlook by the doctor is of inestimable help to patient and friends, though never must it be forgotten by either side that we are dealing with a serious disease.

The price that the patient must pay for his health is eternal vigilance, and Bushnell's aphorism should be remembered by all: "For tuberculosis we prescribe, not medicine, but a mode of life."

Summary.

1. Having made a diagnosis of "early" pulmonary tuberculosis, endeavour to assess your patient in terms of his recoverability, and to have as the goal in treatment a return to former work or occupation.

2. The old tripod of treatment still stands fast: rest, fresh air and good food. A sanatorium *régime* best fulfils these basic principles.

3. It is important to fit details of treatment to the individual case, with close cooperation between doctor and patient and with the maintenance by both of a healthy optimism.

4. Artificial pneumothorax is being employed more frequently and earlier in pulmonary tuberculosis. In every patient the possibility of its induction should be considered early, not as a last resort.

5. Whatever treatment is adopted, remember there are no "short cuts", and a regulated life must be insisted on and maintained always: "*Vive ut vivas.*"

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COTTER HARVEY, M.B., Ch.M. (Sydney), T.D.D. (Wales),

Honorary Assistant Physician, Royal Prince Alfred Hospital; Honorary Physician in Charge of the Clinic for Pulmonary Diseases, Royal North Shore Hospital of Sydney.

Correspondence.

THE SIGNIFICANCE OF ORAL SEPSIS IN THE ADULT.

SIR: In reading Dr. H. C. Adams's paper in the January 28 issue of your journal, on "The Significance of Oral Sepsis in the Adult", one is impressed by various factors: Firstly, the necessity of the constant reiteration of home truths before they are generally accepted; secondly, the conservatism or reluctance of people to accept a new doctrine; thirdly, the length of time required for this acceptance; and, finally, the stormy path the pioneer has to tread who has the temerity to voice such. It has been thus throughout the ages and will ever be so. In writing his paper Dr. Adams is fully alive to the necessity of driving home these truths, as he, like myself, must have been constantly impressed by the number of cases treated by his colleagues of good standing in whom the primary causation of the patient's disease has been overlooked; but he will escape the adverse criticism of his predecessors, as all will heartily agree with him. Emerson says in his article on "Self Reliance": "Great works of art have no more affecting lesson for us than this. They teach us to abide by our spontaneous impression with good-humoured inflexibility then most, when the whole cry of voices is on the other side. Else tomorrow a stranger will say with masterly good sense what we have thought and felt all the time and we shall be forced to take with shame our own opinions from another." Whilst working for some years as clinical assistant and later as acting out-patient physician at the Melbourne and out-patient physician to Saint Vincent's Hospital, I was greatly impressed by the remarkable improvement which took place in patients who had their infections removed, and in March, 1920, published a paper entitled "Chronic Disease and Its Association with Focal Infections", which gave statistics of nearly a thousand cases and showing that about 65% of these cases were suffering from diseases attributable to these infections. Other papers referring to infections and disease were: March, 1919, "The Toxic Syndrome"; November 29, 1919, "Cardiac Affections"; April 19, 1920, a letter maintaining that iritis was the result of infection; August 10, 1918, "A Plea for the Recognition of the Part Played by Focal Infections in the Production of Shell Shock"; May 9, 1925, "Sinusitis as a Source of Disease and Its Effective Treatment", and various others in *The Medical Journal and Record* of New York. The contents of these papers were the results of clinical observation and experiment and were being substantiated by the work done by Rosenow in America, though in many cases they went much further. These, on their publication, expressing such contrary views to the generally accepted opinions of the time, met with a very hostile reception. A paper published by Dr. G. P. O'Day on January 6, 1923, entitled "The Tonsil Considered by a General Practitioner", whilst giving a very scathing criticism of these views, served a very useful purpose, as it expressed the general opinion at that time. Needless to say that in trying to forward views which one knew to be true, one was always in conflict with dentists and rhinologists, as each was satisfied in his own department that his views were right, and it often led to great

difficulties in inducing the patient to follow the right course. It would be foolish to expect that 100% of one's ideas and clinical results would pan out accurately; but time has already shown the truth of the majority, and it will yet show the truth of many more.

The understanding that these focal infections are responsible for so many diseases, only limited by the number of varying tissues of the body, is one of the most revolutionary events that has taken place in the history of medicine, perhaps only rivalled by the views of Lister. Holding views contrary to those generally accepted has the effect of causing others to look upon that individual as some poor harmless creature with a bee in his bonnet, and it often requires a good deal of courage to press one's views to get results, especially when public professional opinion holds that so-called indiscriminate enucleation of tonsils and extraction of teeth was uncalled for; so many took the more conservative way of leaving them alone and allowing their patient to quietly drift into further ill-health rather than get a name for such things. I hope I shall be pardoned for bringing forward these matters, but when one sees one's life's work on the verge of being accomplished, and as there are now likely to be numbers of papers published on this subject, it did not seem out of place to mention these facts and so justify one's original contentions.

The advice given by Isopell Berners to Romany Rye in the Dingle, that when dissension arises, was to say: "God have mercy on my soul and tip in Long Mitford", is always worth bearing in mind.

Yours, etc.,

S. Pern.

12, Collins Street,
Melbourne, C.1,
February 14, 1933.

AN ECONOMIC COMMENTARY AND DIAGNOSIS.

Sir: Objectors to the discussions under the heading "An Economic Commentary and Diagnosis" surely fail to realize that there is a potent interrelation between the body human, the body economic, and the body spiritual, and that it is appropriate that diagnostically inclined minds should be applied to defects in any principle affecting these bodies. There is no doubt concerning the financial future of the profession if the present economic condition persists unrectified. It can only go on till income will not pay professional costs of upkeep of the majority of the profession. The present world-wide upset has a humanly engineered basis, but that I shall not here discuss. What I do maintain is that the present condition of things is not a class problem and is not in reality, as is commonly put forward, a quarrel between the "Haves" and the "Have-Nots". It is a problem which affects everyone, and increasingly so, and serious students insist that on the outcome of this crisis rests the future of our world. Sir Basil Blackett and Sir H. Bowden have, as openly as they can, warned us all quite recently of the possibility of impending catastrophe. Major Clifford Hugh Douglas has been so warning Britons for the past fourteen years. In "Planned Money" Sir Basil Blackett says:

> The twentieth century is now painfully discovering that the *laissez-faire*, self-regulating economic system of the nineteenth century is a myth and that the marvellous material prosperity which the world has enjoyed in the seventy-five years preceding 1914 was not due to the supposed merits of the system, but to a fortuitous combination of circumstances, essentially precarious and ephemeral, which has now passed away. It has taken a war unparalleled in history to drive home the lesson that some better method must be found and applied to the ordering of man's economic life than the unregulated competitiveness of human beings, guided by their own self-interest, however enlightened.

Whilst firmly believing that Dr. Gerald O'Day, in advancing the plan of Russian communism as the way out, is sincere, honest and humane in his endeavours to aid humanity, I have the belief, amounting almost to knowledge, that this method is wrong for us. The problem for Russia, presented in grossly distorted light as it is by the general Press, is nevertheless at the present time, and for a long time to come, fundamentally and radically different from the problem confronting western civilization. Russia's problem is one of actual scarcity; ours is diametrically the opposite one of glut. When Russia reaches the stage which it has taken western civilization two hundred years to arrive at, she will arrive at the problem in front of us. It cannot be too clearly understood that we cannot at present, in the nature of things, learn anything from Russia. Dr. O'Day is probably more entitled to respect than many of our elected representatives, who should know better and who, instead of considering intensely new methods advanced, are busy anæsthetizing the public mind in favour of those who wish to maintain the present impasse as it is.

It is a challenge to the intelligence of the medical and other learned professions to fearlessly and unbiasedly sit in on this question. The London *Times* of November 3, 1932, in a leading article, says that our expert and professional economists have failed us and will not find the solution. The solution will come, outside these, from minds not prejudiced by preconceived ideas.

The hush-hush attitude of those in power concerning the invulnerable, comprehensive, logical and mathematical analysis and the untamperable proposals advanced by Major C. H. Douglas, is probably a most significant suggestion that they will get us out. He conclusively points out the errors in the present methods of industrial loan-cost accountancy which are the main basic, but not the only faults in our financio-distributing system, and his proposals for cure not only close the ever-widening gap between total prices and public ability to buy, but also fulfil the necessity for coping with the ever-decreasing need for so much human labour in the purely economic edifice. The proposals, moreover, are in no way subversive of anything national, for they could be put into effect without any shock to our existing social structure and involve no confiscation, no interference with private property or ownership, no nationalization of industry or of pure banking. As regards the medical profession, the incubus of "L.O.G." and public hospital work would pass away; for the people would then have the money with which to pay. Today they have not got the money, and that is the fact. Accounting figures or money, of which notes and coins are really only the looseleaf parts of the credit ledger books, do not by a long way exist in fact, to measure up to the goods and services available. I would suggest an earnest study of Douglas's own books. What amazes one most is that once their economic truths and import are grasped, the correlation between his economic possibilities and the spiritual possibilities expounded by the Galilean two thousand years ago is astounding. Whereas Communism is anti-Christ and anti-God, C. H. Douglas's proposals are the practical complement to our religious teachings.

Yours, etc.,

"M.B., Ch.B."

February 18, 1933.

Sir: Dr. O'Day's letter of the 18th instant requires an answer, but a very brief one, from me.

If communism is such a success in Russia, how is it that no one outside of Russia wishes to get inside, but a considerable number inside the haven desire to get out of it?

If the single tax were adopted in any country I personally would hurry there on the wings of the wind.

Yours, etc.,

Paul G. Dane.

110, Collins Street,
Melbourne,
February 20, 1933.

DIATHERMY OF TONSILS.

SIR: Two facts stand out in the world-wide discussions on the tonsil problem: (i) that from 50% to 75% of tonsillectomies are inefficiently performed, (ii) that the only settled benefit is the conferring of immunity to diphtheria. The barrenness of post-operative results is no doubt largely due to number one.

I started my career when very few tonsillectomies were ordered; then came a long list of conditions for which it was ordered. The time has come when the profession should revise and cut down that list considerably. In children much more can be done by hygienic measures and exercises than is attempted at present, and if the sinusitis, which is almost invariably present, be treated with diathermy a very large number of operations will be avoided.

A year ago a boy of seven was brought to me; he was pale and listless, and had a distressing hacking cough, headache and frequent colds in the head. He was taken away from school for three months, given exercises, heliotherapy and diathermy. He improved quickly. When he went back to school he had a slight set-back. Today he is quite free from all his troubles, and his tonsils are only slightly enlarged. Not many years back I would have considered removal of tonsils and adenoids the only rational procedure, and I am sure he would have had a long period of convalescence.

I think the clinical teachers of the hospitals are to blame in that very few residents and much fewer students are given an opportunity of realizing that tonsillectomy is a difficult job. They see a master perform, and think it is easy, or they watch a poor attempt and consider they can do it just as well.

I know the curriculum is overloaded, but I would urge that during the anatomy course some attention should be given to the tonsil and nasal sinuses. They are much more important in everyday practice than minute anatomy. Also, during their hospital course the importance and frequency of nasal sinusitis should be drummed into students, not only by specialists, but by physicians and surgeons.

It is no help for surgeons to belittle electrocoagulation of tonsils and to condemn it because someone has been careless or clumsy. Dr. Graham Brown admits it may be perfect and painless. Its only drawback is that it is occasionally somewhat tedious. A year ago I asked anyone who came across a case of mine that was unsatisfactory to publish the fact. After eight years it speaks volumes for electrocoagulation that no one has brought one forward. There are plenty of people who do not understand either diathermy or one who would be only too glad to make a failure known.

Yours, etc.,

W. KENT HUGHES.

22, Collins Street,
Melbourne,
February 20, 1933.

SIR: It is to be regretted that Dr. Bettington should open up the controversy on this subject, especially when he admits that he has had only a small experience with the method. Had his experience been much more mature and his opinions based upon a considerable amount of practice in the use of diathermy, he would, I feel sure, have directed his energies towards advising would-be diathermists of the long and patient training necessary to effect an efficient technique providing for unquestioned results. That he fails to do this is but an indication of the brief practice which he has had with diathermy. He states that most throat specialists have written against the method; but this again can only be taken as a censure of these men, because there is indisputable evidence that the method is sound and effective from men whose status in the ear, nose and throat world does not compare unfavourably with that of our own respected specialists. It is to the credit of the older men practising this specialty that with one or two exceptions they have refrained from joining issue on the question. They surely must have seen bad results and most of them have tried the method, usually with poor success, because too much was expected in far too short an application, yet they were too generous to judge on the poor work of others and on their own very limited experience.

It is not a bit of use Dr. Bettington's talking about extreme pain during and after operation and of infective remnants, and of others speaking of hæmorrhage and peritonsillar damage. If throat surgeons find these results, then with respect I must say it is they and their technique, not the method, which is at fault, and I myself cannot claim any wizardry. I have now done about 300 cases which means nearly 2,000 applications, and the results obtained in nearly all cases have been very gratifying. Only once have I seen a secondary hæmorrhage and this but a slight ooze which was stopped in five seconds with the diathermy spark without any anæsthetic. Pain is a rare symptom and if disabling is the fault of the operator and usually caused through touching the tonsillar pillars. I have not known a patient to miss a meal nor to remain away from work, and I make careful inquiries. If peritonsillar damage is caused this surely can only be the fault of the operator. Certainly some of my early efforts were not successful, as behold the following case. The patient, a man, received about five applications of the diathermy needle after which I was convinced that his symptoms were not referable to his tonsils. I asked him to keep in touch with me over a period of twelve months, but heard later that he had consulted an ear, nose and throat surgeon who advised removal of his tonsils. The day following the operation this surgeon, carrying a tray with the tonsils deposited thereon, walked to the patient's bedside and dramatically exclaimed: "Here are your tonsils; take these to Dr. Smith." This incident proved a blessing, for I realized that no matter what opinion one may have on the question of leaving tonsillar remnants, no business founded on such a basis could possibly succeed, and I promptly set to work to remove all vestige of tonsils that could be found in every case. Only one man of the 300 cases was impossible to complete because he was unable to tolerate the tongue depressor.

The ear, nose and throat specialist has no more right to arrogate to himself the privilege of removing tonsils by diathermy because he is a throat surgeon, than has the general practitioner because he, perhaps, has more experience in the general use of the machine. Nor do I hold that the anatomy of the tonsil is shrouded in mystery and difficulty and but a sanctuary for declining and budding ear, nose and throat specialists. On the contrary, I believe that the treatment of oro-nasal sepsis should be the first and foremost requirement demanded of every general practitioner, and I for one cannot leave undefended an attack upon a method which is eminently successful with a minimum amount of discomfort, but very trying in application and demanding infinite patience and a sound techinque.

Yours etc.,

F. WILKIE SMITH.

Rosebery.
February 20, 1933.

Proceedings of the Australian Medical Boards.

TASMANIA.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Act*, 1918, of Tasmania, as duly qualified medical practitioners:

Brown, Peter Frederick Russell, M.B., B.S., 1928 (Univ. Melbourne), Queenstown.

Bottrell, Henry Reginald Stanley, L.S.A., 1892 (London), L.R.C.S., 1905 (Edinburgh), Flinders Island.

Parker, Douglas William Leigh, M.B., Ch.M., 1923 (Univ. Sydney), F.R.C.S., 1925 (Edinburgh), M.Ch.Orth., 1930 (Liverpool), Bellerive.

Stiles, Bernard Tarleton, M.B., B.S., 1906 (Univ. Sydney), Launceston.
Abey, Arthur Montague, M.B., B.S., 1932 (Univ. Melbourne), Public Hospital, Launceston.
Mitchell, Douglas Harold, M.B., B.S., 1926 (Univ. Melbourne), F.R.C.S., 1931 (England), Launceston.

Obituary.

WILLIAM BRADRIDGE STUDDY.

We regret to announce the death of Dr. William Bradridge Studdy, which occurred on March 9, 1933, at North Sydney, New South Wales.

Books Received.

THE HISTORY OF DERMATOLOGY, by W. A. Pusey, A.M., M.D., LL.D.; 1933. Springfield (Illinois): Charles C. Thomas. London: Baillière, Tindall and Cox. Demy 8vo., pp. 236, with illustrations. Price: $3.00 net.

GROWTH, by the late James Lorrain Smith, M.D., LL.D., D.Sc., F.R.S.; Edited by J. S. Haldane, C.H., M.D., F.R.S.; 1932. Edinburgh: Oliver and Boyd. Demy 8vo., pp. 142. Price: 6s. net.

Diary for the Month.

MAR. 21.—New South Wales Branch, B.M.A.: Medical Politics Committee.
MAR. 22.—Victorian Branch, B.M.A.: Council.
MAR. 24.—Queensland Branch (B.M.A.): Council.
MAR. 28.—New South Wales Branch, B.M.A.: Council (quarterly).
MAR. 30.—South Australian Branch, B.M.A.: Branch.
MAR. 30.—New South Wales Branch, B.M.A.: Annual Meeting.
MAR. 30.—New South Wales Branch, B.M.A.: Annual Meeting.
APR. 3.—New South Wales Branch, B.M.A.: Organization and Science Committee.
APR. 4.—New South Wales Branch, B.M.A.: Council.
APR. 5.—Victorian Branch, B.M.A.: Branch.
APR. 5.—Western Australian Branch, B.M.A.: Council.
APR. 6.—South Australian Branch, B.M.A.: Council.
APR. 7.—Queensland Branch, B.M.A.: Branch.
APR. 11.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
APR. 18.—New South Wales Branch, B.M.A.: Ethics Committee.

Medical Appointments.

Dr. V. M. Plummer (B.M.A.) has been appointed Resident Medical Officer at the Mareeba Babies' Hospital, South Australia.

* * *

Dr. F. A. Bouvier has been appointed, pursuant to the provisions of the *Workers' Compensation Act*, 1928, to be Certifying Medical Officer at Dimboola, Victoria.

* * *

The undermentioned have been appointed resident medical officers at the Perth Hospital, Western Australia: Dr. N. G. Row, Dr. P. Thomas, Dr. J. M. Flynn, Dr. A. A. Merritt, Dr. R. Greenham, Dr. H. C. Robjohns, Dr. E. R. Beech, Dr. K. Clemens (pathologist).

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", page xvi.

MATER MISERICORDIÆ CHILDREN'S HOSPITAL, BRISBANE, QUEENSLAND: House Physician, House Surgeon.
ALFRED HOSPITAL, MELBOURNE, VICTORIA: Honorary Officers.
AUSTIN HOSPITAL FOR CHRONIC DISEASES, HEIDELBERG, VICTORIA: Sanatorium Medical Officer.
GOVERNMENT OF TONGA: Chief Medical Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Mount Isa Mines. Toowoomba Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing. Lower Burdekin District Hospital, Ayr.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	Combined Friendly Societies, Clarendon and Kangarilla districts. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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